IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Applicants: HEDMAN et al.	Art Unit: 3643
Serial No.: 10/014,727	
Filed: December 10, 2001	Examiner: Kurt C. Rowan
Title: METHOD OF KILLING ORGANISMS AND REMOVAL OF TOXINS IN ENCLOSURES	

DECLARATION OF SEAN ABBOTT UNDER 37 C.F.R. § 1.132

Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

1. I, the undersigned, am President of Natural Link Mold Lab. I have a Ph.D. in Biological Sciences from the University of Alberta. I also earned an M.S.C. in Mycology and B.S.C. in Zoology from the University of Alberta. My Curriculum Vitae is also attached as Exhibit A to this declaration. I regularly test sites treated by the Thermal Pest Eradication (TPE) process that includes air filtration, which is described by the above referenced patent application, by examination of air and surface samples to determine levels of contamination by fungi and bacteria.

2. This declaration is submitted in response to the Examiner's rejection of claims 18-23, 26-30, 36-40, and 42-43 under 35 U.S.C. § 103(a) as obvious in view of Forbes and Montellano in the Office Action mailed February 7, 2006. I believe that the obviousness of the claims is rebutted by the long-felt need for the invention.

3. TPE Associates presently licenses and trains pest extermination and environmental services companies in the process of TPE under the name

ThermaPureHeat™ that is covered by the claims of the patent application. As will be further described below, the TPE process uses heat to destroy active mold growth sites and kill viable mold spores, bacteria, viruses, insects, and other heat-sensitive pests and organisms and then filters the heated circulating air. Filtering the air to remove fine particulate matter as part of the eradication process solves the long-felt problem of particulate mass in the air following heat remediation and, thus, the TPE process is not obvious.

4. The need for a way to treat buildings, structures, and other enclosable areas contaminated by mold, bacteria, termites, dust mites, and other microorganisms has been long-felt. Traditional methods to treat buildings contaminated by these organisms are insufficient and may actually create a corresponding problem of increased bioaerosol particulate matter.

5. For example, the traditional method to eradicate pests by tenting a building and filling it up with toxic gas for a period of time sufficient to kill pests, leaves behind dead organisms, which may continue to cause health problems, in addition to the more well known drawbacks of this treatment. These include the requirements of significant amounts of time to be effective and that food and medication must be sealed off or removed. Entire buildings must be treated even if the infestation is localized. Additionally, this method does not eradicate all organisms including bacteria, mold, and certain insects.

6. Similarly, the traditional thermal eradication method, described by the Forbes patent, kills wood-destroying insects like termites by applying a heated gas to wooden surfaces until the surface is heated to a temperature about 120°-135° F, which effectively kills termites. This method, however, is not effective for killing other organisms such as fungi and toxic molds. Further, many insects are serious health hazards, even when dead, and Forbes does not disclose a method for removing the remaining particulates that can actually be resuspended into the air by the injection of heated air into the building. Thus, there is a

continuing need for methods to treat contaminated buildings and deal with the increased aerosol particulate matter found in remediated buildings.

7. Several publications attached to this declaration also support the long-felt need. The first is an article by M.P. Fabian et al. titled "Ambient bioaerosol indices for indoor air quality assessments of flood reclamation." Aerosol Science 36 (2005) 763-83 (Exhibit B). This paper in the Journal of Aerosol Science reports the results of an air quality study that was conducted in residences that were cleaned and reoccupied following a major regional flood. The study used several air-quality indices to assess the effects on common flood reclamation practices on indoor quality. Both indoor and outdoor air quality was sampled after the flooding occurred and after remediation efforts. The remediation efforts included wetted carpets being replaced, soaked dry walls and subfloors being patched or replaced, surfaces washed with bleach, and forced-air dryers applied.

8. Most of the homes damaged by flooding had higher concentrations of airborne particulate matter indoors than outdoors, based on both optical counting (OPC) of airborne particulate matter and composite observations of volatile organic components (VOC). "These results are the opposite of bioaerosol concentration trends typically observed in houses with no water damage." The air samples collected in the houses reclaimed from flood damages also had significantly higher airborne microorganism levels than in houses with no flood damage.

9. The Fabian paper shows that filtration of air following heat remediation is not obvious because it describes the problem of poor air quality following traditional remediation methods. The invention is not obvious in light of this peer-reviewed publication because it reveals the need for filtration of air to remove particulate matter following heat remediation. In my expert opinion, if the invention were obvious, the study would consider the effects of air filtration and would likely suggest it as being part of the heat remediation process. This paper shows the non-obviousness of the invention because it describes the problem of increased

bioaerosol contaminants resulting from typical flood damage clean-ups and does not allude to nor suggest using air filtration in conjunction with the drying out process described.

10. Another pertinent paper is by Ralph E. Moon, Ph.D. CHMM, CIAQP, titled "Thermal Treatment: Benefits and Misconceptions of Using High Temperature Heat (>120° F)" (Exhibit C). This paper discusses the microbial impacts, health and safety consequences of high temperature (120°-160° F) heating and notes that "Even after the living organism is dead, fungal spores, mycelia and mycotoxins still pose an allergenic concern." Page 2. "Turbulent fans assist the drying process; however, they also aerosolize microbial matter and dust. As a result, turbulence also creates potential combustible conditions by the emancipated dust." Pages 10-11.

11. The Moon paper is relevant because one of the safety concerns it addresses is dust, which is solved by the filtration step of the TPE process. This paper shows the non-obviousness of the invention because it describes the long-felt need for the invention by disclosing the safety concerns of dust and the fact that even dead microorganisms can be allergens, but does not suggest filtration.

12. An article by Alan Forbess "Heat Treatment Method Provides Water Damage/Mold Relief," Claims May 2006 (Exhibit D) discusses the process of using heat to destroy organisms and the pitfalls of standard mold remedy which is costly and consuming. The Forbess paper shows the non-obviousness of the invention because it describes the long-felt need for the invention and its usefulness, especially in terms of cost-saving.

13. The April 2006 issue of Cleaning Specialist Q&A by Jim Holland describes heat remediation (Exhibit E). The Holland paper describes hot-air drying in general and then explains the problem of sewage damage. In discussing the considerations of using heat remediation the author notes that it is "important to

remember that even though elevated temperatures kill pathogens, the organic material remains and may result in major odor problems over time." With regard to using heat to assist in mold remediation, the author claims that killing mold spores "probably would still not be an acceptable technique for treating mold in crawlspace since the 'dead organisms' are still problematic." The paper also quotes "The medical effects of mold exposure" by R.K. Bush et al in the Journal of Allergy and Clinical Immunology, Volume 11,7 number 2, pages 326-33 (Exhibit F), "Allergic responses to inhaled mold antigens are a recognized fact in lower airway disease (i.e., asthma)." Also quoting that article, hypersensitivity pneumonitis "is an uncommon but important disease that can occur as a result of mold exposure." These conditions can both result from dead spores killed by heat, but left in the building. In my opinion, this paper shows the non-obviousness of the invention because it describes the long-felt need uniquely solved by this invention and does not recognize the use of air filtration in conjunction with the heat remediation process.

14. "Airborne Particle Sizes and Sources Found in Indoor Air" by M.K. Owen et al. in the Atmospheric Environment, Vol 26A, No 12 pp 2149-2162 (1992) (Exhibit G). This peer-reviewed paper looks at the indoor aerosols including the mechanics of deposition in the lungs and the dynamics that influence particles. The article surveys information about indoor aerosols, particularly particle sizes. This paper is pertinent to the case at hand because it discusses the health implications of inhaling indoor aerosols. "Bioaerosols, including bacteria and viruses, present special health hazards due to the risk of infection." Page 2149. The paper describes aerosol formation and explains that resuspension, large solid particles reentering the air, can occur with sweeping or in-breezes. My opinion is that this paper shows the non-obviousness of the invention because it describes the impact of bioaerosol particulate matter, which, as discussed above, is increased in concentration after traditional remediation efforts.

15. The TPE process described in the patent application addresses this long-felt need by effectively killing organisms in enclosures, eliminating substantially all such organisms, in a manner that is non-toxic, and can be performed in a relatively short time, is clean, dry and odorless, and removes a large proportion of the dead organisms. The invention solves a long-felt unresolved need, and therefore one may infer that the invention is nonobvious. If it were obvious, someone would have previously developed the invention to solve the need.

16. As a microbial expert, I am well aware of the other processes that are available for eradication of organisms. Based upon the testing I have done and my own familiarity with the industry, I am of the opinion that, prior to the introduction of the ThermaPureHeat™ process, no process or service existed that solved the problem of eradicating contamination, in particular filtering the air to collect the airborne particulate matter left behind after traditional eradication methods. The failure of others to determine a solution for the long-felt need assists in establishing that the solution was nonobvious.

17. In light of the long-felt need for a method to safely and effectively eradicate contaminants from buildings, I believe that the claims of the patent application are not obvious in view of the prior art cited in the Office Action dated February 7, 2006.

18. I hereby declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code and that such willful false statements may jeopardize the validity of the application or any patent issued thereon.

Dr. Sean Abbott, Ph.D.

6/7/06
Date

CURRICULUM VITAE

SEAN P. ABBOTT

revised November 08, 2005

Academic Record/ Education

2000	Ph.D.	(Fungal Systematics) Dept. of Biological Sciences, University of Alberta, Edmonton, AB, Canada
1992	M.Sc.	(Mycology) Dept. of Botany, University of Alberta, Edmonton, AB, Canada
1989	B.Sc.	(Specialization Zoology) Dept. of Zoology, University of Alberta, Edmonton, AB, Canada

Awards/ Scholarships

Name of Scholarship or Award	Agency or Institution	Date
Myron Backus Award	Mycological Society of America	1999
Luella K. Weresub Award	Canadian Botanical Association	1998
John Macoun Travel Bursary	Canadian Botanical Association	1998
Izaak Walton Killam Memorial Scholarship	The Killam Trusts	1998 – 2000
Andrew Stewart Memorial Graduate Prize	University of Alberta	1998
NSERC Postgraduate Scholarship (PGS-B)	Natural Sciences and Engineering Research Council of Canada	1996 - 1997
Walter H. Johns Graduate Fellowship	University of Alberta	1996 - 1997
Challenge Grants in Biodiversity	Alberta Conservation Association And U of A Biological Sciences	1996 – 1998
NSERC Postgraduate Scholarship (PGS-1&2)	Natural Sciences and Engineering Research Council of Canada	1989 - 1991
Walter H. Johns Graduate Fellowship	University of Alberta	1990 - 1991
Northern Science Training Grant	Boreal Institute for Northern Studies	1989 - 1990
Graduate Faculty Fellowship	University of Alberta	1989 - 1990
NSERC Undergraduate Student Research Award	Natural Sciences and Engineering Research Council of Canada	1988
Undergraduate Scholarships (2)	Province of Alberta	1986 - 1988
Alexander Rutherford Scholarships (2)	Province of Alberta	1981 - 1983

Summary of Employment Experience

Nov 2001 – present

President, Natural Link Mold Lab, Inc., July 2004 - present (formerly Analytical Director, akaMOLDLAB, Nov 2001 – July 2004), 390 Freeport Blvd. Unit 3, Sparks, NV 89431.

- Oversee mycology/microbiology laboratory, primarily centered on indoor fungal contamination and IAQ.
- Responsible for maintaining laboratory quality and integrity of analytical procedures.
- Teaching and training; classes workshops, presentations and curriculum development for training programs of private and public groups as well as internal company staff training.
- Consulting for industry organizations for development of reliable processes and practices in the field of environmental microbiology.
- Legal consulting/expert witness for various law firms nationwide.
- R&D; efficacy testing of new technologies and processes, antifungal product testing, project consultation, etc.

Jan 2000 - Oct 2001

Director of Analytical Operations (Mar 2001)/ Senior Mycologist, Environmental Microbiology Laboratory, Inc., 1800 Sullivan Ave., Suite 209, Daly City, California 94015 (D. Gallup, President).

- Oversee operations of a rapidly expanding analytical laboratory in the field of indoor fungal contamination and aerobiology. Responsible for maintaining high quality mycological analysis and helping to set industry standards in this new and growing field of environmental mycology.
- First full-time analyst at Daly City lab (Jan 2000), Laboratory Manager (May 2000).
- During tenure as Laboratory Manager/Director, increased volume (gross monthly revenue) by 600%. Growth achieved by matching industry growth through aggressive hiring and training of quality personnel, improving client support and education, expansion of services, evaluation of costs and streamlining of analytical processes.

Aug. 1998 - Dec 1999

Director, Novobios Ltd.

- one-third partner of small business involved with biological technologies and consulting, including fungal biodiversity inventories, microbial contamination assessment and orchid micropropagation.

Jan 1996 - Dec 1999

Research Associate/Consultant, Casual, University of Alberta Microfungus Collection and Herbarium (L. Sigler, Professor) and U of A Devonian Botanic Garden (D. Vitt, Director).

- consulting projects involving airborne molds as biological hazards, indoor microbial contamination, fungal identification (UAMH) and orchid collection management and horticulture (DBG).

Feb 1992 - Dec 1995

Mycologist/Research Associate, University of Alberta Microfungus Collection and Herbarium (L. Sigler, Professor).

- research projects on fungal systematics, anamorph-teleomorph connections of Ascomycetes and Basidiomycetes, airborne molds as biological hazards, opportunistic human pathogens, etc.
- experience with maintenance of a living culture collection.

Jan 1989 - Sep 1989

Research Assistant, University of Alberta Devonian Botanic Garden (R. Currah, Professor).

- research projects on zoophilic Ascomycetes, holomorph studies of *Cystoderma*, Boletaceae and Cantharellaceae flora of Alberta.

May 1988 - Sep 1988

Research Assistant, University of Alberta Devonian Botanic Garden (R. Currah, Professor).

- taxonomy of Alberta ectomycorrhizal fungi.

May 1987 - Sep 1987 Research Assistant, University of Alberta Devonian Botanic Garden (R. Currah, Professor).
- floristics of Alberta Ascomycetes.

May 1986 - Sep 1986 Seasonal Horticulturalist, University of Alberta Devonian Botanic Garden (G. Ford, Assistant Director).
- horticulture and floristics of native Alberta vascular plants.

Academic Teaching

1999	BOT 306	Biology of the Fungi (R. Currah), 1 lecture
1998	MMI 427	Medical Mycology (L. Sigler), 3 lectures
1998	BIOL 108	Introductory Biology (R. Currah), 1 lecture
1998	BOT 306	Biology of the Fungi (R. Currah), 1 lecture
1997	BIOL 108	Introductory Biology (R. Currah), 1 lecture
1996	MMI 427	Medical Mycology (L. Sigler), 2 lectures
1995	BOT 306	Biology of the Fungi (R. Currah), 2 lectures
1994	MMI 427	Medical Mycology (L. Sigler), 1 lecture
1992	MMI 427	Medical Mycology (L. Sigler), 1 lecture
1992	BOT 380	Drug Plants of the World (K. Denford), 1 lecture

Laboratory Demonstration:

1991	BOT 306	Biology of the Fungi (R. Currah)
1990	BOT 306	Biology of the Fungi (R. Currah)
1989	BOT 406	Biology of the Fungi (R. Currah)

Extension Teaching and Public Education

2001-present Continuing Education (CE) credits provided for courses, workshops and presentations given for various agencies, including:
American Board of Industrial Hygiene (ABIH)
Board of Certified Safety Professionals (BCSP)
California Association of Medical Laboratory Technicians (CAMLT/CLS)
California Certified Laboratory Scientist (CA CLS)
Continuing Education Center For Pest Management (DPR)
Nevada Association of Real Estate Inspectors (NACREI)

2002-2003 Senior instructor and microbial director for mold courses taught by National Environmental, Inglewood, CA, including *Mold Remediation* and *Mold Inspector* courses.

1987-1999 I have taught over 45 courses and workshops on various aspects of biology, including mushroom identification, native plant identification, ornithology, natural history, orchid biology, etc. These courses have been offered through many agencies including:
- Devonian Botanic Garden: *Mushroom Identification* (1989, 1990, 1995, 1996, 1997, 1998), *Hunting for Edible Mushrooms* (1991, 1992, 1993, 1994, 1996, 1997), *Spring Mushrooms* (1990, 1991, 1992, 1993, 1994, 1995, 1996, 1998), *Fall Mushrooms* (1992), *Intermediate Mushroom ID* (1993), *Identifying Alberta Wildplants* (1991), *Discovering Spring Birds* (1996), *Natural History of the Sandhills at the Devonian Botanic Garden* (1995), *Orchids Galore* (1999), *Orchid Horticulture* (1997, 1998, 1999).
- University of Alberta Extension: *Mushroom Identification* (sessional lecturer 1991).
- John Jansen Nature Centre: *Mushroom Mania* (spring and fall 1991, 1992).
- Powerhouse Continuing Education: *Identifying and foraging for mushrooms* (1996).
- Ukrainian Cultural Centre: *Spring Mushrooms* (1999).

- Alberta Agriculture: *Introductory Mushroom Identification Workshop* (1987, 1988).
- Edmonton Mycological Society: *Mycology/Botany Field Course* (1989), *Mushroom Identification* (1988).
- Fort McMurray Natural History Club: *Mushroom Identification Workshop* (1996).
- Alberta Native Plant Council: *Mushrooms of the Devonian Botanic Garden* (1995, 1996).
- Stony Plain Horticultural Society: *Introductory Mushroom Identification Workshop* (1988).
- Individual Study Course: *Micropropagation of Orchids from Seed* (1998, 1999).

1997-1999 Devonian Botanic Garden Master Gardener's certificate course. Invited to teach half day sessions on *Orchid Horticulture*, lectures and demonstration of techniques of micropropagation and orchid growing. (5 courses; Jul. 1997, Nov. 1997, Mar. 1998, Nov. 1998, Mar. 1999).

1987-present I have given lectures for various interest groups including:

- University of Nevada Reno, Environmental Science and Health Seminar Series: *Fungi In The Indoor Environment* (2004).
- University of Alberta Devonian Botanic Garden: *Species conservation and orchid horticulture in Thailand* (1998); *Poisonous mushrooms and toxic molds* (1996); *Fungi at the DBG* (1995).
- Edmonton Mycological Society: *Mushroom growing and natural history in northern Thailand* (1998); *False Morels* (1996); *Fungal photography* (1991); *Spring Ascomycetes* (1990), *Mycorrhizal and species-specific edibles* (1988).
- Alberta Wilderness Sportsman Club: *Edible and Poisonous Mushrooms of Alberta* (1999).
- Foothills Orchid Society (Calgary): *Orchid species conservation in Thailand* (1999).
- Orchid Society of Alberta: *Orchid conservation in Thailand* (1998).
- Master Gardener's Club: *Growing Orchids* (1997).
- University of Alberta. of Botany: *Taxonomic studies of the Helvellaceae in northern and northwestern North America* (1992).
- BOT 1000, The U of A Dept. of Botany: *A fungal feature* (1990).
- U of A Wildlife, Wildlands Seminar Series: *Mushrooms and other fungi of Alberta* (1990).
- John Jansen Nature Centre Young Naturalists: *Mushrooms and fungi* (1990).

Publications (in refereed journals)

Sime, A.D., L.L. Abbott and S.P. Abbott. 2002. A mounting medium for use in indoor air quality spore-trap analyses. Mycologia 94:1087-1088.

Abbott, S.P., T.C. Lumley, and L. Sigler. 2002. Use of holomorph characters to delimit *Microascus nidicola* and *M. soppii* sp. nov., with notes on the genus *Pithoascus*. Mycologia 94: 362-369.

Abbott, S.P. and L. Sigler. 2001. Heterothallism in the Microascaceae demonstrated by three species in the *Scopulariopsis brevicaulis* series. Mycologia 93: 1211-1220.

Lumley, T.C., S.P. Abbott, and R.S. Currah. 2000. Microscopic Ascomycetes isolated from rotting wood in the boreal forest. Mycotaxon 74: 395-414.

April, T.M., S.P. Abbott, J.M. Foght, and R.S. Currah. 1998. Degradation of hydrocarbons in crude oil by the ascomycete *Pseudallescheria boydii* (Microascaceae). Canadian Journal of Microbiology 44: 270-278.

Abbott, S.P., L. Sigler, and R.S. Currah. 1998. *Microascus brevicaulis* sp. nov., the teleomorph of *Scopulariopsis brevicaulis*, supports placement of *Scopulariopsis* with the Microascaceae. Mycologia 90: 297-302.

Abbott, S.P. and R.S. Currah. 1997. The Helvellaceae: systematic revision and occurrence in northern and northwestern North America. Mycotaxon 62: 1-125.

Sigler, L. and S.P. Abbott. 1997. Characterizing and Conserving diversity of filamentous basidiomycetes from human sources. Microbiology and Culture Collections 13: 21-27.

Abbott, S.P., L. Sigler, and R.S. Currah. 1996. Delimitation, typification, and taxonomic placement of the genus *Arachnomyces*. Systema Ascomycetum 14: 79-85.

Sigler, L., S.P. Abbott, and H. Gauvreau. 1996. Assessment of worker exposure to airborne molds in honeybee overwintering facilities. American Industrial Hygiene Association Journal 57: 484-490.

Currah, R.S., S.P. Abbott, and L. Sigler. 1996. *Arthroderma silverae* sp. nov. and *Chrysosporium vallenarense*, keratinophilic fungi from arctic and montane habitats. Mycological Research 100: 195-198.

Abbott, S.P., L. Sigler, R. McAleer, D.A. McGough, M.G. Rinaldi, and G. Mizell. 1995. Fatal cerebral mycoses caused by the ascomycete *Chaetomium strumarium*. Journal of Clinical Microbiology 33: 2692-2698.

Sigler, L., S.P. Abbott, and A.J. Woodgyer. 1994. New records of nail and skin infection due to *Onychocola canadensis* and description of its teleomorph *Arachnomyces nodosetosus* sp. nov. Journal of Medical and Veterinary Mycology 32: 275-285.

Abbott, S.P. and R.S. Currah. 1988. The genus *Helvella* in Alberta. Mycotaxon 33: 229-250.

Publications (books)

Abbott, S.P. and R.S. Currah. 1989. *The larger cup fungi and other ascomycetes of Alberta.* University of Alberta, Devonian Botanic Garden, Edmonton. 96 Pp.

Non-refereed Publications (published abstracts, conference proceedings, technical reports, theses)

Chase, L., D. Hedman and S.P. Abbott, 2005. Thermal remediation: A new application of an old process. Facility Safety Management October: 20-23.

IICRC (S.P. Abbott, contributing author). 2003. IICRC Standard and reference guide for professional mold remediation S520. Institute of Inspection, Cleaning and Restoration Certification, Vancouver, WA.

Abbott, S.P. 2002. Microbial contamination in HVAC systems. The Construction Zone 2(9): 9.

Abbott, S.P., D.H. Vitt, and L. Sigler. 2002. Ex-situ conservation of orchids and orchid mycorrhizal fungi at the Devonian Botanic Garden. Proceedings of the 16th World Orchid Conference. Vancouver Orchid Society, Vancouver. Pp 429.

Abbott, S.P. 2002. Sampling for airborne molds. The Construction Zone 2(8): 23.

Abbott, S.P. 2002. Mycotoxins and Indoor Molds. Indoor Environment Connections 3(4): 14-24.

Abbott, S.P. 2000. Holomorph studies of the Microascaceae. Ph.D. Thesis, University of Alberta Dept. of Biological Sciences, Edmonton. 196 Pp.

Abbott, S.P. 1999. Diversity of decay fungi in boreal habitats. The bio-diversity grants program biennial report 1997/98, University of Alberta, Edmonton, P. 3.

Abbott, S.P. 1999. Orchid source book: a procedures manual for maintenance of the orchid and epiphyte collection and display house. University of Alberta Devonian Botanic Garden, Edmonton. 125 Pp.

Sigler, L., P.C. Kibsey, D.A. Sutton, S.P. Abbott, E. Zilkie, D.I. McCarthy, and A. Fothergill. 1999. *Monascus ruber,* causing renal infection. Abstracts, American Society for Microbiology, 99th annual meeting, Chicago. Pp. 297.

Abbott, S., I. Johnston, L. Sigler, and D. Vitt. 1999. Ex-situ conservation of orchids and orchid mycorrhizal fungi at the Devonian Botanic Garden. Abstracts, 16th World Orchid Conference, Vancouver. Pp 29.

Sigler, L., S.P. Abbott, and R.C. Summerbell. 1998. Comment on the correspondence between Dr J. Guarro and Dr C. Rajendran in *Medical Mycology* 1998; 36: 349-50. Medical Mycology 37: 79.

Abbott, S.P., L. Sigler, and R.S. Currah. 1998. Holomorph studies of the Microascaceae: Disparate relationships of *Scopulariopsis brevicaulis* and *Scopulariopsis canadensis*. Abstracts, 34th annual meeting of the Canadian Botanical Association, Saskatoon. Pp. 50.

Sigler, L. and S.P. Abbott. 1998. Airborne mold analysis and microbial assessment of four schools in SW British Columbia. University of Alberta Microfungus Collection and Herbarium, Edmonton. 34 Pp.

Abbott, S.P. 1997. Diversity of decay fungi of the family Microascaceae in boreal and montane habitats. The bio-diversity grants program biennial report 1996/97, University of Alberta, Edmonton. Pp. 49-50.

Sigler, L., S.P. Abbott, and J. Frisvad. 1996. Rubratoxin mycotoxicosis by *Penicillium crateriforme* following ingestion of home-made rhubarb wine. Abstracts, 96th general meeting of the American Society for Microbiology, New Orleans. F-22, Pp. 77.

Sigler, L. and S.P. Abbott. 1996. Filamentous basidiomycetes from clinical sources. In: *Culture collections to improve the quality of life* (Samson et al. eds.). Proceedings of the eigth International Congress for Culture Collections, Veldhoven. Centraalbureau voor Schimmelcultures, Baarn, Netherlands and World Federation of Culture Collections. Pp. 386-389.

Abbott, S.P., L. Sigler, R. McAleer, and D. McGough. 1995. Fatal cerebral mycoses caused by *Chaetomium strumarium*. Abstracts, 95th general meeting of the American Society for Microbiology, Washington DC. F-128, Pp. 109.

Gauvreau, H., L. Sigler, and S.P. Abbott. 1995. Assessment of airborne molds as a biological hazard for Alberta commercial beekeepers. Alberta Occupational Health and Safety, Edmonton. 72 Pp.

Abbott, S.P. and L. Sigler. 1994. Arthroconidial anamorphs of basidiomycetes. Abstracts, fifth International Mycological Congress, Vancouver. Pp. 1.

Abbott, S.P. and L. Sigler. 1994. Filamentous basidiomycetes from clinical sources. Abstracts, XII congress of the International Society for Human and Animal Mycology, Adelaide, Australia. PO5.29, Pp. D133.

Abbott, S.P. 1992. Systematic studies of the Helvellaceae in northern and northwestern North America. M.Sc. Thesis, University of Alberta Dept. of Botany. 174 Pp.

Abbott, S.P. and R.S. Currah. 1991. Evolutionary trends towards a hypogeous existence seen in ascomycetous fungi from Alberta. Abstracts, Canadian Botanical Association, Edmonton. No. 50, Pp. 34.

McDonald, D., R.S. Currah, and S.P. Abbott. 1991. Zoophilic ascomycetes from Svalbard including a new genus and species in the Arthrodermataceae. Abstracts, Canadian Botanical Association, Edmonton. No. 53, Pp. 35.

Currah, R.S., L. Sigler, A. Flis, and S.P. Abbott. 1989. Cultural and taxonomic studies of ectomycorrhizal fungi associated with lodgepole pine and white spruce in Alberta. University of Alberta Microfungus Collection and Herbarium, Devonian Botanic Garden, Edmonton. 100 Pp.

Abbott, S.P. and R.S. Currah. 1988. The genus *Helvella* in Alberta with special emphasis on a new species in the section *Acetabulum*. Abstracts, Canadian Botanical Association, Victoria. Pp. 82.

Invited Presentations

Sep. 25, 2005	Invited speaker for Connections Convention and Trade Show, Las Vegas, NV. Lecture: *Microbiology of the Indoor Environment.*
Jul. 12, 2005	Invited speaker for ThermaPureHeat-WaterOut Symposium, Phoenix, AZ. Lecture: *Microbial Aspects of Thermal Remediation.*
Mar. 11, 2005	Invited speaker for American Indoor Air Quality Council (AmIAQ), Santa Barbara Chapter, Ventura, CA. Lecture: *Microbial Issues Other Than Mold Affecting Indoor Air Quality.*
Feb. 17, 2005	Invited speaker for Environmental Solutions Association (ESA), First Annual Conference, Atlantic City, NJ. Lecture: *Microbiology Sampling Methods and Data Interpretation.*
Jun. 4, 2004	Invited speaker for Precision Environmental/ThermaPure, Ventura, CA. Lecture: *Why Heat? Microbial Aspects of Thermal Remediation.*
Oct. 24, 2003	Invited speaker for American Indoor Air Quality Council (AmIAQ), Santa Barbara Chapter, Inaugural Meeting, Santa Barbara, CA. Lecture: *S-520 – The New IICRC Standard for Mold Remediation: A Status Report.*
Jul. 31, 2003	Invited speaker for USDA Rural Development, Multi-family Housing Owners & Managers Policy Meeting, Carson City, NV. Lecture: *Fungi In Indoor Environments.*
June 14, 2003	Invited speaker for American Institute of Architects, Burbank, CA. Lecture: *Mold: The Causes, The Consequences, The Cure And "Cooking The Mail.*
May 17, 2003	Invited speaker for Nevada Association of Certified Real Estate Inspectors (NACREI), Reno, NV. Lecture: *Investigationg Fungal Contamination in Indoor Environments.*

Nov. 21, 2002	Invited speaker for the Association of Applied IPM Ecologists, 2nd School and Urban IPM Workshop, Elk Grove, CA. Lecture: *Integrated Pest Management: Control of Insects and arthropods vectors of sporedispersal in fungi.*
Nov. 6, 2002	Invited speaker for American Indoor Air Quality Council, Los Angeles, CA. Lecture: *The HEAT is on: Insects and other arthropods as agents of vector-dispersal in fungi.*
Oct. 24, 2002	Invited speaker for Ventura County Coastal Association of Realtors, Oxnard, CA. Lecture: *Mold contamination in buildings: Health, legal and remedial Issues. Welcome to the kingdom Fungi.*
Oct. 16, 2002	Invited speaker for National Parks Service, Yosemite, CA. Lecture series on Integrated Pest Management. Lecture: *Insects and other arthropods as agents of vector-dispersal in fungi.*
Sep. 12, 2002	Invited speaker for Apartment Association Southern California Cities, Long Beach, CA. Lecture: *Fungi in indoor environments.*
July 21, 2002	Invited speaker for California Association of Medical Laboratory Technicians (CAMLT), Sparks, NV. Lecture series co-presented with Dr. Nancy McClenny: Lecture: *Environmental Mycology.*
July 18, 2002	Invited panel member for CM/WLS Roundtable, Sacramento, CA. Served as mycology/microbiology expert for panel discussions.
May 18, 2002	Invited speaker for Nevada Association of Certified Real Estate Inspectors (NACREI), Reno, NV. Lecture: *Investigationg Fungal Contamination in Indoor Environments.*
May 4, 2002	Invited speaker for American Society for Microbiology Northern California Chapter (NCASM), Santa Clara, CA. Lecture series co-presented with Dr. Deanna Sutton: *A day of mycology.*
Apr. 10-11, 2002	Invited speaker for Western Regional Conference on Lead, Mold, Healthy Homes and Children's Environmental Health, Berkeley, CA. Lecture: *Strategies and Tools for Conducting Environmental Assessments;* Chair of *Technology Demonstrations and Discussions* session for the *Workshop on Developing, Managing and Financing a Healthy Homes Program.*
Oct. 19, 2001	Invited speaker for American Industrial Hygiene Association Southern California Chapter, Long Beach, CA. Lecture: *Mold/Fungi sampling techniques, data interpretation and guidelines.*
July 13, 2001	Invited speaker for Rocky Mountain Center for Occupational and Environmental Health, University of Utah, Denver, CO. Lecture series: *Indoor Mold Issues: an overview.*
Apr. 20, 2001	Invited speaker for Rocky Mountain Center for Occupational and Environmental Health, University of Utah, Salt Lake City, UT. Lecture series: *Indoor Mold Issues: an overview.*
Apr. 19, 2001	Invited speaker for IHI Environmental, Salt Lake City, UT. Lecture: *The biology of indoor molds.*
Apr. 6, 2001	Invited speaker for Safe Environments & Precision Works, Inc., San Mateo, CA. Lecture: *Mold contamination in buildings: health, legal and remedial issues.*

Mar. 29-30, 2001	Invited speaker for American Industrial Hygiene Association Arizona Chapter, Phoenix, AZ. Lecture series co-presented with Dr. Phil Morey: *Advanced course in bioaerosol investigations.*
Sep. 27, 2000	Invited speaker for the Environmental Law Forum, San Francisco, CA. Lecture: *Indoor air quality (IAQ), moisture intrusion and microbial amplification.*
May 9, 2000	Invited speaker for SINA Environmental Educational Seminar: Mold & Fungi, Dublin, CA. Lecture: *Mold growth in indoor environments.*
Feb. 22, 2000	Invited speaker for Benchmark Environmental Training, Mold and Indoor Air Quality Seminars, Freemont, CA. Lecture: *Implications of fungal growth in the indoor environment.*
Mar. 27-28, 1998	Invited speaker for Woodlot Association of Alberta, Non-timber Forest Products Workshop at Olds College, Olds, AB. Lecture: *Mushrooms as a non-timber alternative.*
June 4-6, 1993	Invited lecturer for Alberta Natural Areas Volunteer Steward Conference, hosted by the Natural and Protected Areas Section of Alberta Environmental Protection, at Seebee, AB. Lecture: *Mushrooms of Alberta.*

Additional Training

2005	Susceptibility Testing and Non-Fermentor ID (Gram-negative bacteria), workshop by P. Schreckenberger, sponsored by the Hardy Diagnostics, Palm Springs, CA, January 21, 2005.
2000	Identification of common *Penicillium* species, workshop by J. Pitt sponsored by the National Laboratory Training Network, New Orleans, LA, April 11-15, 2000.
1999	Commercial Horticulture - Tissue Culturing, course attended through the U of A Devonian Botanic Garden Education Programme.
1996	Radiation Safety Course, certificate received from Radiation Control Committee, Occupational Health and Safety, University of Alberta.
1995	Digital Microscopy & the Internet and Applications of the Variable Pressure SEM Workshop, hosted by Nissei Sangyo Canada and Surgical-Medical Research Institute, University of Alberta.
1994	Biological Scanning Electron Microscopy Course, certificate received from Surgical-Medical Research Institute, University of Alberta.
1993	Transport of Dangerous Goods/IATA Training Course, certificate received from Biosafety Officer, Occupational Health and Safety, University of Alberta.

Other Evidence of Scholarly and Creative Achievement

May 6, 2004-present	Invited participant of IICRC S520 Revision Committee, inaugural meeting, San Diego, CA. Invited to join IICRC S520 Committee as chair of *Fungal Ecology Committee,* co-Chair of *S520 Glossary Committee,* and member for the *Health Effects Committee* and *Tools, Equipment, Materials Committee*: Preparation and review of final draft of revised S520 publication of fungal remediation standard.
May 4, 2003-Dec 2003	Invited participant of IICRC S520 Committee meeting, Las Vegas, NV. Invited to join IICRC S520 committee member for *Fungal Ecology Committee*: Preparation and review of final draft of S520 publication of fungal remediation standard.
March 2003-present	Invited to provide scientific consultation for the Nevada Senate Subcommittee regarding pending mold-related legislation (SB 131, 132 in 2003; AB 303 in 2005).
Jan 2002-present	Serve as Chair of the *Basic Science Committee* of the Indoor Environmental Institute (IEI).
Jan 2003-present	Serve on the Founding Advisory Board for the American Air Quality Council (AmIAQ), Santa Barbara Chapter.
Feb 23-26, 2001	Visiting scientist at the Centers for Disease Control (CDC), Atlanta, GA. Study of laboratory techniques and cooperative discussions with Dr. B. Fields and staff regarding testing of environmental samples for the presence of *Legionella* bacteria.
Dec 2000	Served as peer referee for grant application submitted to The National Science and Engineering Research Council of Canada (NSERC), application for operating funds for individual scientist/university professor.
Jul 22, 1999	Received *Certificate of Appreciation for Outstanding Volunteer Service in Horticulture* from the University of Alberta Devonian Botanic Garden for assistance with establishment and maintenance of the orchid collection (1998/1999).
Apr 26-May 1, 1999	Attended World Orchid Conference, Vancouver. Presented conservation poster (see publications). Served as conference co-organizer for the micropropagation demonstrations which provided the public an opportunity to learn about the complex laboratory requirements of orchid propagation from seed.
Dec 15-31, 1997	Invited visiting scientist and lecturer at Maejo University, Chiang Mai, Thailand. Cooperative studies between the U of A Devonian Botanic Garden and Maejo University Faculty of Agricultural Production. Areas of focus included conservation of botanical biodiversity, orchid horticulture, and public education and awareness of natural history. • Final Report (Abbott, S.P. 1998. University of Alberta Devonian Botanic Garden - Maejo University Dept. Horticulture Linkage Project) submitted to U of A Dept. Rural Economy, Faculty of Agriculture, Forestry and Home Economics, and the Canadian International Development Agency (CIDA).
Mar 26, 1997	Ph.D. Candidacy Examination: *Pass with commendation.*
1987-1999	I served as a registered consultant for the Alberta Poison Control Centre for cases of mushroom poisoning in central and northern Alberta and NWT.
1992-present	Served as peer reviewer for manuscripts submitted to Mycologia, Medical Mycology, The Bryologist, Canadian Journal of Botany, Mycological Research, and Kew Bulletin.

1987-1999

Founding member of the Edmonton Mycological Society and executive officer (Editor 1987-1991; Program Coordinator 1996-1999).

1980-present

Various articles submitted for newsletters and amateur publications including:
The Kinnikinnick (Friends of the Devonian Botanic Garden): 8 articles including:

- Abbott, S.P. 1999. The 16th World Orchid Conference. Kinnikinnick 14 (2): 7-8.
- Abbott, S.P. 1999. The orchid house: settling in and spreading the word. Kinnikinnick 14 (1): 1-3.
- Abbott, S.P. 1998. The epiphytic garden: evolution of the orchid house. The Kinnikinnick 13 (2): 1-4.
- Abbott, S.P. 1997. A new orchid collection. The Kinnikinnick 12 (2): 1-3.
- Sigler, L. and S.P. Abbott. 1996. Homemade rhubarb wine health alert. The Kinnikinnick 11 (3): 6-7.
- Abbott, S.P. 1996. The incredible *Stanhopea* orchid. The Kinnikinnick 10 (1): 9-10.
- Sigler, L. and S.P. Abbott. 1995. Furnace fungi and indoor molds - Fungi to be feared? The Kinnikinnick 9 (3): 8-9.

Orchid Society of Alberta Newsletter: articles and notes including:

- S.P. Abbott. 1999. The epiphytic garden: a brief history of the Orchid House at the Devonian Botanic Garden. 38 (3): 4-5.

The Foothills Orchid Society Newsletter: articles and notes including:

- S.P. Abbott. 1999. The epiphytic garden: a brief history of the Orchid House at the Devonian Botanic Garden. April 1999: 5-6.

The Stinkhorn (Edmonton Mycological Society): Vols. 1-5, over 20 articles including:

- Abbott, S.P. and L. Abbott. 1991. The Alberta *Verpa* report. The Stinkhorn 5 (1): 13-18.
- Abbott, S.P. 1991. Pet poisonings. The Stinkhorn 5 (1): 22-23.
- Abbott, S.P. 1991. Book reviews - Mushrooms of western Canada. The Stinkhorn 5 (1): 41.
- Abbott, S.P. 1988. The genus *Helvella*. The Stinkhorn 2 (1): 12.
- Abbott, S.P. 1987. Foray at the Botanic Garden. The Stinkhorn 1 (1):14-15.
- Abbott, S.P. 1987. *Pholiota squarrosa* poisoning. The Stinkhorn 1 (1): 24.

The Mycelium (Toronto Mycological Society): 1 article.

- Abbott, S.P. 1982. *Russula* poisoning. The Mycelium 8 (5): 3.

1990-present

Interviewed for newspaper articles in *The Construction Zone* (Las Vegas, NV), *The Edmonton Sun* (Edmonton, AB), *The Edmonton Journal* (Edmonton, AB), *The Devon Dispatch* (Devon, AB), *Folio* (Edmonton, AB), *The Gateway* (Edmonton, AB), *The Morinville & District Gazette* (Morinville, AB), *St. Albert Gazette* (St. Albert, AB). Topics include mold contamination, wild mushrooms in Alberta, fairy ring mushrooms, snow mold, orchids, etc.

Interviewed for television: KTVN Channel 2 News (Reno, NV); CFRN news (Edmonton, AB); ITV First news (Edmonton, AB); Channel 10 (Edmonton, AB) and radio: CBC 'The Good Question' (Edmonton, AB); CBC (Calgary, AB) regarding fungi, bacteria and orchids.

1995-1999

Coordinator of "Mushroom Magic", an annual event highlighting mushrooms and other fungi, aimed at increasing public awareness of fungi, hosted by the Devonian

	Botanic Garden and Edmonton Mycological Society.
1995/96, 1998	Coordinator of Devonian Botanic Garden Seminar Series.
Sep 5-8, 1996	Schalkwyk Conference, Fungi of Western Canada, Conference Organizer (Foray Coordinator, Jasper National Park Liaison and Transportation Assistant Coordinator). Presented paper: *Helvellaceae of Western Canada.*
Jun 23-27, 1991	Helped with organization of Canadian Botanical Association meeting in Edmonton, served on Volunteer Committee as chair of 'Registration Packages' group and as projectionist.
1987-1991	Editor of *The Stinkhorn* (Edmonton Mycological Society), Volumes 1-5, a publication of amateur mycology in Alberta comprising articles on Alberta mushrooms, species lists, mycological news, illustrations by local artists, etc.
Jan-Feb 1991	Visiting student at the Mycological Herbarium, Royal Botanic Gardens, Kew, England (B. Spooner/ D. Pegler) and the International Mycological Institute, Kew, England (B. Sutton/ P. Cannon/ J. Pryce). • Independent study examining collections of Helvellaceae.

Memberships in professional and scientific societies:

- American Conference of Governmental Industrial Hygienists (2002-2005)
- American Indoor Air Quality Council (2001-present)
- American Industrial Hygiene Association (2001-present)
- American Orchid Society (2001-present)
- American Society for Microbiology (2002-present)
- Edmonton Mycological Society (1987-1999)
- Indoor Air Quality Association (2002-present)
- Indoor Environmental Institute (2002-present)
- Mycological Society of America (1996-present)
- North American Mycological Association (1981-present; life member)
- Orchid Society of Alberta (1998-1999)
- Orchid Society of Northern Nevada (2003-present)
- Pan American Aerobiology Association (2002-present)
- Toronto Mycological Society (1981-1984)

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Ambient bioaerosol indices for indoor air quality assessments of flood reclamation

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Abstract

An air quality study was conducted in arid-region residences that were cleaned and reoccupied following a major regional flood (Arkansas River, Colorado, USA). This demonstration study leveraged a suite of aerosol measurements to assess the effects of common flood reclamation practices on indoor air quality. These assays included (i) optical counting (OPC) of airborne particulate matter (0.3–5 μm optical diameter), (ii) composite observations of volatile organic compounds (VOC), (iii) culturing and direct microscopic counts of airborne bacteria and fungi, and (iv) air-exchange rate measurements. As judged by OPC, most of the flood damaged homes surveyed had higher concentrations of airborne particulate matter indoors than outdoors; the same trend was observed for selected VOC. When compared to large literature databases, culturing from air samples collected in houses reclaimed from flood damage had significantly higher airborne microorganism levels than in houses where no flood damage had occurred—in many cases this difference was between two and three orders of magnitude. As determined by direct epifluorescence microscopy, total airborne microorganism concentrations were 3–1000 times higher than those recovered by conventional culturing. In flood damaged homes, biological particles averaged 52% of the total particles measured indoors, and 18% of the total particles measured immediately outdoors. Relative differences between the indoor and outdoor concentrations of airborne particulate matter, microorganisms, and associated VOCs, suggested that flood-impacted building materials were sustaining high aerosol bioburdens and

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contributing to poor indoor air quality more than 3 months after the structures had been reclaimed from flood damage.

Keywords: Bioaerosol; Indoor air quality; Fungi; Bacteria; Flood

1. Introduction

Poor indoor air quality has been shown to cause adverse health effects. While air quality indices and exposure levels are well defined in terms of certain chemical compounds and particulate matter, they are poorly defined regarding airborne contaminants of microbiological origin. As a generic class of airborne pollutants, particulate matter usually associated with compounds of biological origin is often termed "bioaerosol". This definition includes all airborne microorganisms regardless of viability or ability to be recovered by culture; it comprises whole microorganisms as well as fractions, biopolymers and products from all varieties of living things (ACGIH, 1999). Indoor bioaerosols can originate from outdoor air or from internal sources such as building occupants and their activities, and building materials that host microbiological growth.

Numerous indoor air quality publications report that airborne biological particles range in aerodynamic diameter between 0.01 and 100 μm (ACGIH, 1999). In many indoor environments, airborne bacteria, fungi and their fragments may fall into a respirable size range that can penetrate deep into human lungs (< 10 μm) (Górny et al., 2002; Reponen, Grinshpun, Conwell, Wiest, & Anderson, 2001). Higher respiratory morbidity and allergic complaints have been observed in occupants of mold-colonized structures in several studies (Brunekreef et al., 1989; Dales, Zwanenburg, Burnett, & Franklin, 1991; Platt, Martin, Hunt, & Lewis, 1989; Strachan, 1988; Verhoeff & Burge, 1997; Verhoeff, van Wijnen, & van Brunekreef, 1995). High airborne bacteria concentrations have also been positively correlated to adverse respiratory symptoms (Björnsson et al., 1995). However, bioaerosol concentrations responsible for adverse health effects have not been defined.

Airborne bacteria and fungi can be toxigenic, allergenic and/or infectious. While only complete microorganisms can be infectious, toxic and allergic reactions can be caused by microorganism fragments or byproducts (Burrell, 1991; WHO, 1990). Examples include endotoxin, a compound found in Gram-negative bacteria cell walls (ACGIH, 1999); microbial volatile organic compounds (VOC), products of bacterial and fungal metabolism (ACGIH, 1999; Miller, 1992); β-(1–3)-D-glucans, found in fungal cell walls (ACGIH, 1999); and mycotoxins, products of fungal metabolism (Robbins, Swenson, Nealley, Gots, & Kelman, 2000). Cell and spore fragments can be important sources of allergens and toxins, as their numbers can be several magnitudes higher than cells or spores released from building materials, depending on the species, environmental conditions and wind velocity (Górny et al., 2002).

Fungal and bacterial growth, in and on water-damaged building materials, is a potential health hazard and many recent reports contain evidence to support this observation (Abe & Nagao, 1996; Bardana, 2003; Zureik et al., 2002). The incidence of human disease has been reported to increase markedly following the flooding of residential areas (Marwick, 1997; MMWR, 1993a, b, 1994). While some of these diseases can be traced to waterborne infectious agents and to conventional disease vectors (i.e. mosquitoes), many cannot be linked to specific sources. In this context, there is relatively little information regarding aerosol

exposures within flood damaged residences to suggest an epidemiological link between exposure and adverse health outcomes.

The literature concerning human bioaerosol exposures and associated regulatory limits is tenuous. At present, neither the US Environmental Protection Agency (EPA) nor the National Institution of Occupational Safety and Health (NIOSH) have proposed concentration limits for bioaerosols. One of the earliest guidelines was proposed in 1946 which suggested that no more than 0.1–20 colony forming units (CFU)/ft^3 should grow in 24 h in operating theatres (Topley, 1955). The American Conference of Governmental Industrial Hygienists (ACGIH) reported interim indoor bioaerosol exposure guidelines based on culturable levels of bacteria and fungi, but these guidelines have been repealed since 1999. Those guidelines recommended that less than 100 CFU/m^3 was an acceptable level (ACGIH, 1989). The Health and Welfare department in Canada proposed the following guidelines: (1) 50 CFU/m^3 of one species of fungi warrants immediate investigation; (2) the presence of certain fungal pathogens is unacceptable; (3) 150 CFU/m^3 of mixed species is normal; and (4) up to 500 CFU/m^3 is considered acceptable if the species present are primarily *Cladosporium* (Environment Canada, 1989; WHO, 1990). The European Union also suggested bioaerosol concentration exposure thresholds in terms of CFU, suggesting guidelines for residential and industrial environments (CEC, 1993). More recently, Górny and coworker reviewed European literature databases on residential indoor air quality and proposed the following residential limit values: 5×10^3, 5×10^3 CFU/m^3, and 5 ng/m^3 for airborne fungi, bacteria and bacterial endotoxin, respectively; the presence of pathogenic fungi is considered unacceptable in any concentration (Górny & Dutkiewicz, 2002). In 1994, the New York City Department of Health issued guidelines for assessment and remediation of indoor fungal contamination. This report qualified recommendations in the context of biological indoor air quality problems with the statement "it is not possible to determine "safe" or "unsafe" levels of exposure..." (NYC-DOH, 1994). To determine the presence of significant indoor microbiological sources, these guidelines also recommended comparisons of the species recovered from standard plate counts in addition to comparing the microorganism concentrations recovered from parallel air samples collected indoors and outdoors. These recommendations have become standard for many other organizations (ACGIH, 1999; WHO, 1990), and an extensive review by Rao and Burge lists many organizations and the guidelines they have presented (Rao, Burge, & Chang, 1996).

Most of these guidelines are based on baseline (bio)aerosol concentrations, without taking into account effects on human health (Rao et al., 1996). In addition, most studies have proposed threshold bioaerosol concentrations based on culturing assays (Reponen, Nevalainen, Jantunen, Pellikka, & Kalliokoski, 1992; Reynolds, Streifel, & McJilton, 1990; Robertson, 1997; Yang, Lewis, & Zampiello, 1993). Organizations such as NATO and WHO have concurred that, there is a need to develop more accurate and robust methods for characterizing biological aerosols (Maroni, Axelrad, & Bacaloni, 1995; WHO, 1990). Since many bioaerosol associated diseases are not dependent upon infection to induce adverse health effects, it is important to quantify all microbial cells that are suspended in the air, as well as differentiating between those that are metabolically active, those that are culturable, and those that are non-viable (Hernandez, Miller, Landfear, & Macher, 1999).

A goal of this demonstration study was to compare common and emerging air quality indices observed in a cohort of single-family residences reclaimed after an arid-region flood, to those observed in non-flood impacted homes. A residential demonstration study was performed in Southern Colorado, USA, where, due to heavy rains, the Arkansas River flooded the city of La Junta. Both indoor and outdoor air was sampled several months after the flooding had occurred and after full-scale remediation efforts, when residents had cleaned and returned to their homes. Novel air sampling paradigms and equipment

were used to determine the total airborne bacteria and fungi concentrations within residences after they were reclaimed from flood damage; these were executed in parallel with conventional culturing assays using non-selective media. These concentrations, together with air-exchange rate monitoring, VOC and airborne particulate matter measurements, were used as evidence to determine if the reclamation efforts following flood damage mitigated the potential for significant microorganism enrichments of indoor air (i.e. higher indoor concentrations).

2. Materials and methods

2.1. Microbiological air quality sampling protocol

The following protocols were applied to monitor building air-exchange rates, airborne microorganism concentrations—both total and culturable—and critical environmental factors in the flood-damaged homes.

Air-exchange rates were estimated using tracer gas tests. Thirty minute monitoring of a CO_2 spike (and its subsequent decay) was executed in the main room of the flood-damaged residences. Following CO_2 tracer tests, bioaerosol samples were collected in swirling liquid impingers (3 h) (Willeke, Lin, & Grinshpun, 1998) and conventional N6 Andersen impactors (1 or 2 min) (Andersen, 1958), while total airborne particle concentrations in the size range between 0.3 and 5 μm optical diameter (OD), were concurrently monitored for up to 4 h. Temperature and relative humidity were recorded hourly during the sampling campaigns.

2.2. Residence selection

Indoor and outdoor air samples were collected and characterized in eight single story flood-damaged houses and one non-flooded house. Building selection was based on similarity in extent of flood damage, the structure (single level), age and construction materials, as well as remediation status (complete). Cleaning was considered complete when wetted carpets had been replaced, soaked dry walls and subfloors had been patched or replaced, non-structural surfaces had been washed with bleach, and forced-air dryers had been applied. Air sampling was executed between 2 and 3 months following their cleaning and reoccupation. This coincided with the summer season, when outdoor bioaerosol concentrations have been implicated as the major source of indoor bioaerosol concentrations in residential buildings (Nevalainen, Pasanen, Reponen, Kalliokoski, & Jantunen, 1991). Passive ventilation (open windows and doors) was the main method used to ventilate these residences when occupied during the summer months.

Residents carried out their normal activities up to a couple of hours before air sampling commenced. Because of the short-term effects of everyday activities on indoor bioaerosol concentrations (Lehtonen, Reponen, & Nevalainen, 1993), there were no human or animal activities in the residences during the sampling campaigns. Special care was taken not to disturb the residences' interiors; this practice was meant to minimize particle reaerosolization and provide for sampling normalization among the residences sampled.

2.3. Environmental monitoring

Temperature and humidity probes (Fisher Scientific, Fullerton, CA) monitored relative humidity and temperature hourly, both indoors and outdoors, during all sampling periods. To minimize temporal

variations, tracer gas studies were executed, and indoor and outdoor air was sampled at the same times, between 9 am and 2 pm, in every residence. Wind speed data and general weather conditions were obtained from a local meteorological station (La Junta Municipal Airport, La Junta, CO).

2.4. Air-exchange rates

Tracer gas tests were used to estimate air-exchange rates of the residences under the conditions monitored; these CO_2 tests were modified from a widely accepted decay method (Kronvall, 1981; Winberry et al., 1993). The protocol for the decay test was as follows: CO_2 gas was injected in the residences, and allowed to mix and accumulate to a level of 5000 parts-per-million (ppm). Once 5000 ppm was reached, CO_2 injection was ceased and the CO_2 concentration was recorded every minute until the gas had reached background levels (typically 800 ppm indoors). Carbon dioxide was used as a tracer because it is a non-reactive gas that is easy to monitor and does not pose a health threat at the concentrations used. CO_2 was measured using a Langan CO_2 probe fitted with a microprocessor for continuous data acquisition (Langan Products, Inc., San Francisco, CA).

Indoor air mixing was facilitated by small household 120 V box fan (33 cm diameter) placed in the rooms sampled. To reduce the potential for spore release from building materials (Górny, Reponen, Grinshpun, & Willeke, 2001; Pasanen, Pasanen, Jantunen, & Kallikoski, 1991), mixing fans were placed in a manner that did not direct airflow towards the walls. Fans were operated according to the following protocol: ON during tracer gas injection and bioaerosol sampling, and OFF during CO_2 monitoring.

2.5. Microbiologically associated volatile organic compounds (MVOC)

Air samples for selected VOC analyses were drawn into a glass tube containing activated carbon media (Air Quality Sciences, Marietta, GA) using a pump (model 224-PCXR8, SKC Inc., Eighty Four, PA) for 4 h at a flow rate of 0.2 L/min, collecting 48 l of air. Tubes were placed approximately 2 m above the ground, hanging vertically from a rack. Care was taken to place tubes away from walls or close to other potential VOC sources. At the end of the sampling period, tubes were shipped overnight on ice and analyzed with a gas chromatograph/mass spectrometer using widely accepted methods (AQS, 1997). Based on the laboratory equipment sensitivity and volume collected, detection limits for the compounds reported were 10 ng/m^3.

2.6. Bioaerosol collection and analyses

2.6.1. Swirling liquid impingers: BioSamplers

Bioaerosol samples were collected using swirling liquid impingers according to accepted methods (Lin et al., 1999, 2000; Willeke et al., 1998) and manufacturer's specifications (BioSampler, SKC Inc., Eighty Four, PA). The efficiency of the BioSampler filled with 20 ml of water is 79% for 0.3 μm particles, 89% for 0.5 μm particles, 96% for 1 μm particles and 100% for 2 μm particles (Willeke et al., 1998). Particle-free, autoclaved 0.01 M phosphate-buffer saline (PBS) containing 0.01% Tween 80 (SIGMA, St. Louis, MO) was used as the collection medium in all impingers. For bioaerosol sampling, three BioSamplers were placed in clusters at least 1 m above the ground, indoors and outdoors. Outdoor samples were located at least 1 m above the ground, several meters away from open doors and windows to minimize the influence from indoor sources. If samplers had to be placed closer to doors, these were kept shut during

the experiments and alternate routes of entry were used to check the indoor samplers. The BioSampler inlets were oriented such that their directions defined the points of an equilateral triangle, which provided multidirectional collection and reduced any near-field sampling effects the impingers may have had on each other. All impingers were connected to a rotary vane-type vacuum pump (model 1023-101Q-G608X, Gast Inc., Benton Harbor, MI) and collected air at a flow rate of 12.5 L/min (SD = 0.7 L/ min). The vacuum pumps were operated for 5 min prior sampling to assure a constant vacuum source. Flow rates were monitored by three 50 L/min capacity flow meters (Gilmont® Instruments, Barrington, IL) and calibrated with a primary standard airflow bubble meter (Gilibrator, Gilian Instrument Corp., Clearwater, FL).

BioSamplers were operated for a minimum of three consecutive hours during which time they collected 2250 L of air. During extended BioSampler operations, the reservoir liquid evaporates, which can lead to collection efficiency reductions from re-aerosolization and particle bouncing (Lin et al., 1999; Willeke et al., 1998; Grinshpun et al., 1997; Lin et al., 1999). To keep collection efficiency constant, a sterile phosphate saline buffer solution was periodically added to maintain the impingers' reservoir volumes at the manufacturer's recommended level of 20 ml. Buffer was prepared and autoclaved in the laboratory, and, as a precaution, was filter sterilized on-site using a Nalgene vacuum bottle fitted with a 0.2 μm pore filter just prior to using. Approximately every 30 min the pumps were turned off and any evaporated capture buffer was quickly replaced by injecting sterile buffer down the impingers' neck. For this study, which was executed in an arid region with low humidity, it was necessary to replace approximately 4 mL (±1 mL) of buffer every half-hour to keep the manufacturer's recommended liquid levels within the impingers' reservoirs. Before sampling, impingers were washed with deionized water and 70% ethanol and autoclaved for 15 min at 121 °C. Immediately after collection, samplers were stored on ice to minimize microorganism growth, and shipped to the University of Colorado environmental microbiology laboratory (within 4 h) where their contents were aseptically diluted for direct microscopy, and transfer onto agar plates.

2.6.2. Microorganism enumeration: culturability assays via liquid capture

A modification of a standard plate count method (Gerhardt, Murray, Wood, & Krieg, 1994) was used to enumerate culturable bacteria and fungi retained in the impinger's liquid. Within 4 h after collection, liquid samples from impingers were cultured on plates inoculate by a spiral dispenser (Spiral Biotech, Inc., Bethesda, MD) according to the manufacturer's recommendations. At least three replicates of each sample were cultured. A comparison of culturable counts determined with the spiral plater, and those determined by standard spread plate methods, showed no significant differences between the recovery of these methods (based on an independent t-test, $\alpha = 0.05$), and that the spiral plater method variability was lower than that of the spread plate method (coefficient of variance (CV) was 5% lower for the spiral plating method, $n = 10$).

For culturing assays, agar plates were prepared up to a week in advance and stored under aseptic conditions. Culture plates were refrigerated at 10 °C prior to use, and care was taken to avoid the drying effects of long exposures to room temperature or direct sunlight. Bacteria were cultured on tryptic soy agar (TSA) (Difco Laboratories, Detroit, MI) including 0.5% cycloheximide (SIGMA, St. Louis, MO) to prevent fungal growth (Schillinger, Vu, & Bellin, 1999). Fungi were cultured on malt extract agar (2% MEA) (Difco Laboratories, Detroit, MI), which is recommended by the American Conference of Governmental Industrial Hygienists (ACGIH) as a non-selective fungal agar (ACGIH, 1999) including 0.05% chloramphenicol (SIGMA, St. Louis, MO) to inhibit bacterial growth (Schillinger et al., 1999).

This broad-spectrum fungal medium has been recommended for determination of building associated fungi (Samson et al., 1994). Once inoculated, bacterial plates were incubated at 37 °C for 14 days, and CFUs counted every 3 days. Fungal media plates were incubated at 25 °C for 14 days and CFUs counted every 3 days.

2.6.3. Microorganism enumeration of impinger reservoir contents: microscopy assays (total microorganism counts)

Epifluorescence microscopic counting was used to enumerate the total numbers of bacteria and fungi (culturable, and non-culturable) captured in impinger samples. For microscopy, cells were stained with Acridine Orange (AO) (Fisher Scientific, Springfield, NJ), a fluorescent stain that non-selectively binds to nucleic acids (Hobbie, Daley, & Jasper, 1977). Samples for total cell counts were stained at a final concentration of 0.001% AO, incubated for 1 min at room temperature, and filtered through a 25 mm diameter black polycarbonate filter with a pore size of 0.2 μm (Poretics, Inc., Livermore, CA). All direct counts were reported based on counts from the average of 10 microscopic fields. Mounted filters were examined under 1000× magnification using a Nikon Eclipse E400 epifluorescence microscope fitted with a mercury lamp and polarizing filters (HBO-100 W mercury lamp; F/TXRD X excitation filter; F/TXRD M emission filter; F/TXRD BS beamsplitter (ChromaTechnology Corp., Brattleboro, VT)). A 24-bit color digital camera (Spot Camera, Diagnostic Instruments, Sterling Heights, MI) captured fluorescent micrographs, which were then viewed and archived using Adobe Photoshop 5.0 software (Adobe Systems, San Jose, CA).

2.6.4. Microorganism enumeration: culturability assays via solid agar capture in Andersen impactors

A one-stage N6 Andersen impactor (Graseby-Andersen Instruments, Smyrna, GA) was used to compare impaction recovery of airborne bacteria and fungi to that obtained using BioSamplers. This stage collects particles with a 50% cut-off aerodynamic diameter (d_{50}) of 0.65 μm. Impactors were connected to a vacuum pump (model 10709, Andersen Samplers Inc., Atlanta, GA), which collected air at 28.3 L/min. Impactor pumps were calibrated using a bubble meter (Gilibrator, Gilian Instrument Corp., Clearwater, FL). Either 28.3 or 56.6 L of air were collected for each sample (1 or 2 min sample time). The impactor equipment was washed and sterilized with 70% ethanol prior to sampling, and the impactor was operated for 30 s with a sterile, HEPA filtered air to purge any microorganisms trapped from previous handling. Blanks were included to verify sterility. Impactors were placed 1.5 m above the floor, more than 3 m from the BioSamplers. One indoor and one outdoor impactor sample was collected in each house.

Agar plates loaded into the impactor were prepared according to manufacturer's recommendations, and media plates were incubated and counted as previously outlined. Colony counts were adjusted with a positive-hole correction factor to account for the possibility of collecting multiple particles through single holes on the Andersen sampler stages (Macher, 1989).

2.6.5. Total particle counts

An optical particle counter (OPC) model 237B (Met One, Pacific Scientific Company, Chandler, AZ) was used to count as a function of size total (biological and non-biological) particles collected both indoors and outdoors. The particle counter was connected to a timer and solenoid valve that switched between indoor and outdoor sampling every minute. Sampling volume was 1.4 L, collected for 30 s at a flowrate of 2.8 L/min. Particle concentrations were recorded in the following size ranges on the basis of

optical diameter: 0.3–0.5, 0.5–0.7, 0.7–1, 1–2 and 2–5 μm. One hundred samples were collected at each residence, 50 indoors and 50 outdoors, over a time frame of 100 min.

3. Results

3.1. Environmental monitoring

During the sampling periods (between 9 am and 2 pm, 5 h for a typical residence), temperatures indoors and outdoors increased, while relative humidity decreased. In the flood-damaged houses, relative humidity indoors varied between 43 and 88%, and outdoors between 31 and 85%. Temperatures varied between 20 and 28 °C indoors, and between 17 and 35 °C outdoors. Within a single observation, the maximum relative humidity variation was ±7% indoors and ±19% outdoors; the maximum temperature variation was ±2 °C indoors and ±3.2 °C outdoors. Wind speed on the days of the monitoring varied between 8.5 and 16 km/h. Based on the CO_2 decay experiments, air-exchange rates in the houses varied between 0.8 and 3.5 air changes per hour (ACH, 1/h).

3.2. Microbiologically associated volatile organic compounds

Selected VOCs were monitored as indicators of fungal metabolism (ACGIH, 1999; AQS, 1997; Miller, 1992; Pasanen, Lappalainen, & Pasanen, 1996). VOC of possible microbial origin (MVOC) were detected in over half of the flooded houses tested. Three alcohols and one ketone were detected in significant concentrations, varying between 70 and 2710 ng/m^3. The most common VOC found was 3-methyl-1-butanol, which has been associated with fungal growth on building materials (AQS, 1997). Other common MVOC found were 2-octen-1-ol, 2-heptanone, and 1-octen-3-ol. Fig. 1 summarizes the type and quantity of MVOC observed in all houses surveyed.

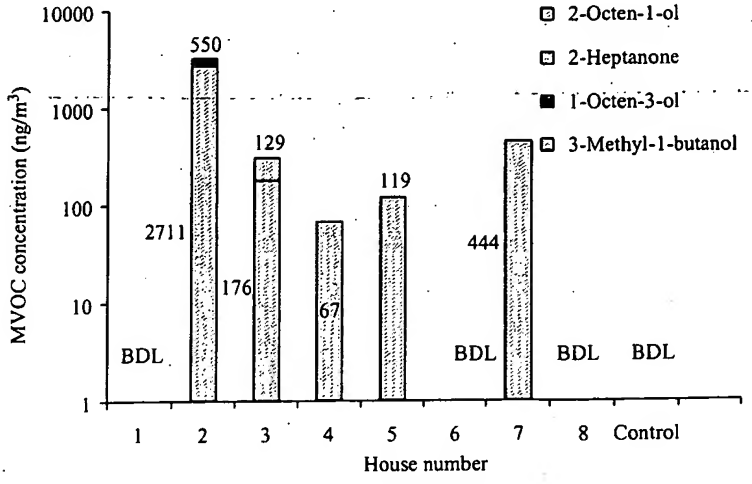
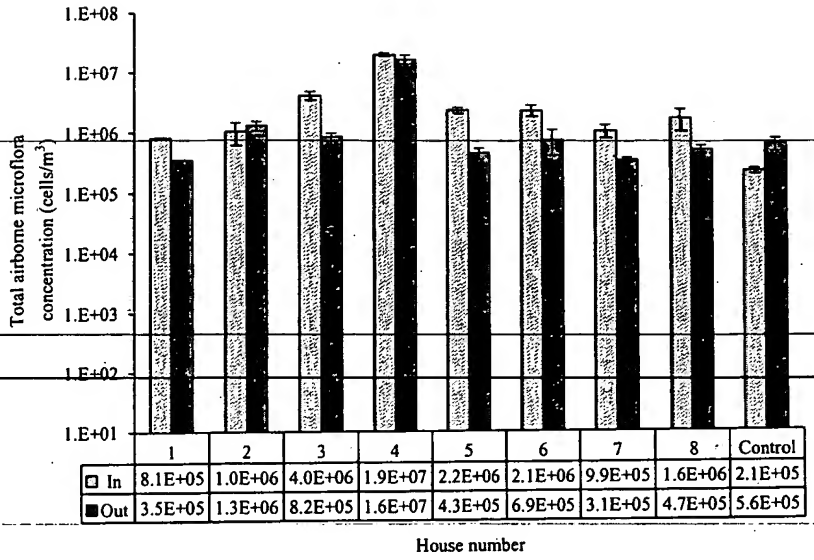


Fig. 1. Type and quantity of microbial volatile organic compound (MVOC) extracted from 48 L of indoor air in flood-damaged and control residences. All outdoor samples collected were below the VOC detection limit. BDL = below detection limit (10 ng/m^3).



	1	2	3	4	5	6	7	8	Control
In	8.1E+05	1.0E+06	4.0E+06	1.9E+07	2.2E+06	2.1E+06	9.9E+05	1.6E+06	2.1E+05
Out	3.5E+05	1.3E+06	8.2E+05	1.6E+07	4.3E+05	6.9E+05	3.1E+05	4.7E+05	5.6E+05

Fig. 2. Average total airborne bacteria and fungi concentrations recovered from SKC swirling liquid impingers in flood-damaged residences, as determined by direct microscopy. Error bars represent one standard deviation, $n = 3$.

3.3. *BioSamplers—total airborne microorganism recovery*

In all flood-damaged houses, total indoor airborne microorganism concentrations ranged between 8.1×10^5 and 1.9×10^7 cells/m^3, and outdoor concentrations ranged between 3.1×10^5 and 1.6×10^7 cells/m^3. Fig. 2 summarizes total airborne microorganism level, as defined by the sum of all bacteria, fungi and spores observed in and near the houses. As judged by t-test at a 95% probability level ($\alpha = 0.05$), seven of eight flooded houses had indoor microorganism concentrations significantly higher than their corresponding immediate outdoor concentrations; one flooded house (house #2) did not show a statistically significant difference between indoor and outdoor total microorganism concentrations, and the local control house had indoor concentrations significantly lower than that measured immediately outdoors. There was a broad diversity of microscopic cellular morphology observed in all the samples collected, and no general trends in morphology were observed. Propagule sizes ranged from less than 1 μm to over 10 μm in diameter. Fig. 3 is an epifluorescence microscope photograph of AO-stained microorganisms typical of those recovered from the air inside flood-damaged houses.

3.4. *SKC liquid impingers—culturable recovery*

3.4.1. *Bacteria*

Mesophilic bacteria were recovered from the SKC liquid impingers on non-selective media (TSA). Seven of the eight flooded houses had higher averages of airborne culturable bacteria concentrations indoors than outdoors (Fig. 4), although only four were statistically different as judged by means and analyses of variance (t-test, $\alpha = 0.05$).

Averages of culturable airborne bacteria recovered from indoor air of flood-damaged homes ranged between 3.9×10^2 and 3.9×10^5 CFU/m^3, while corresponding outdoor concentrations ranged between

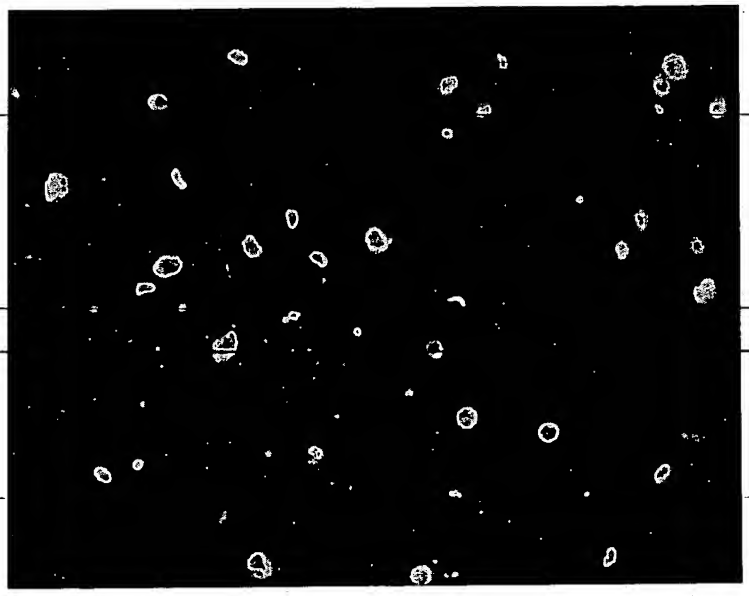
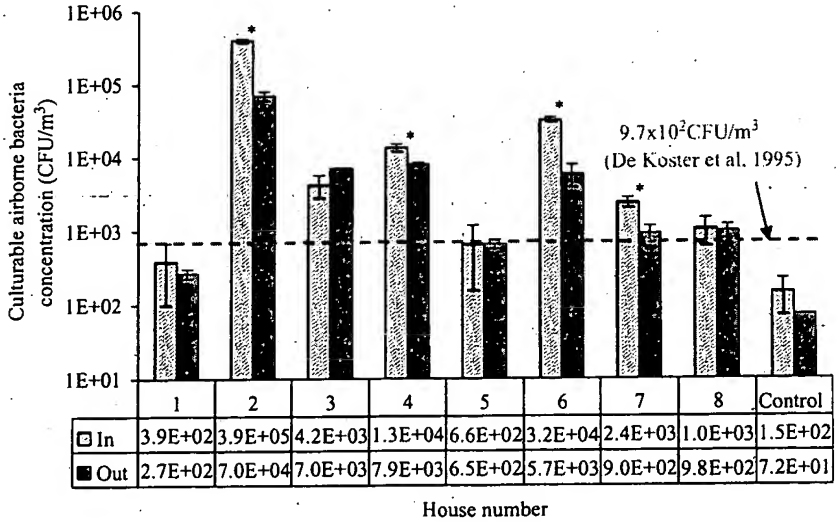


Fig. 3. Epifluorescence microscope photograph of AO-stained bacteria, fungi, and spores collected from the indoor air of a flood-damaged home (1000×).



	1	2	3	4	5	6	7	8	Control
In	3.9E+02	3.9E+05	4.2E+03	1.3E+04	6.6E+02	3.2E+04	2.4E+03	1.0E+03	1.5E+02
Out	2.7E+02	7.0E+04	7.0E+03	7.9E+03	6.5E+02	5.7E+03	9.0E+02	9.8E+02	7.2E+01

Fig. 4. Average airborne concentrations of culturable bacteria recovered from BioSamplers. Error bars represent one standard deviation, $n = 3$. Asterisks denote houses where concentrations were statistically different indoors and outdoors. A line represents the average value of culturable bacteria from a survey of non-flood-damaged US homes, $n = 41$ (DeKoster & Thorne, 1995).

2.7×10^2 and 7.0×10^4 CFU/m^3. The ratios of airborne bacterial concentrations recovered indoors and outdoors varied between 3.5 and 8.8. In a non-flooded residence in the local vicinity, average indoor concentrations were less than 33% of the immediate outdoor concentrations, a ratio which was in agreement with many previous observations (Nevalainen et al., 1991; Samson, 1985; Solomon, 1975; Verhoeff, Brunekreef, Fischer, van Reenen-Hoekstra, & Samson, 1992).

3.4.2. *Fungi*

Impinger-captured aerosol samples were cultured on malt extract agar to maximize the recovery of fungi and their spores. Culturable concentrations of airborne fungi were generally higher indoors than outdoors, and the dominant types of fungal genera cultured from indoor air samples were different from those cultured from outdoor samples. On this non-selective fungal media, four of eight houses had significantly higher culturable concentrations of fungi indoors than outdoors (t-test, $\alpha = 0.05$) (Fig. 5). Average concentrations of culturable fungi from air samples inside flooded houses varied between 1.6×10^3 and 1.0×10^4 CFU/m^3, and immediately outside flooded houses between 5.5×10^2 and 5.0×10^4 CFU/m^3. *Trichoderma* spp. was the colony-forming phenotype most often recovered from indoor air samples, but was not recovered in numerically significant CFUs from any outdoor air samples. *Penicillium* spp. was the colony-forming phenotype most often recovered from outdoor air samples, but was not recovered in numerically significant CFUs from indoor air samples. *Trichoderma* grows optimally in environments with high water activity (Kredics et al., 2004) while *Penicillium* species can grow at a wide range of water activity (Andersen & Frisvad, 2002; Gock, Hocking, Pitt, & Poulos, 2003; Plaza, Usall, Teixidó, & Viñas, 2003). These results indicated that even though the houses had undergone remediation efforts, some building materials were not dry and were promoting the growth of some fungi with an affinity to high water content environments.

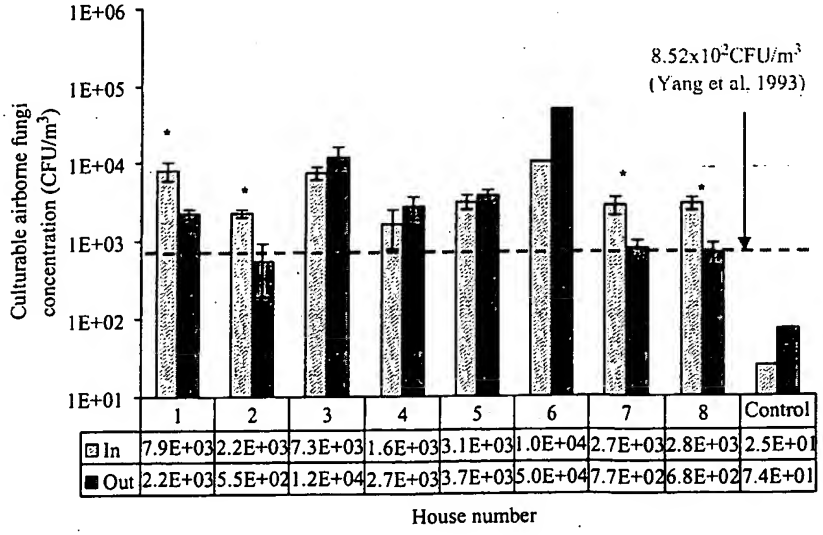


Fig. 5. Average airborne concentrations of culturable fungi recovered from BioSamplers. Error bars represent one standard deviation, $n = 3$. Asterisks denote houses where concentrations were statistically different indoors and outdoors. A line represents the average value of culturable fungi in non-flood-damaged US buildings, $n = 2000$ (Yang et al., 1993).

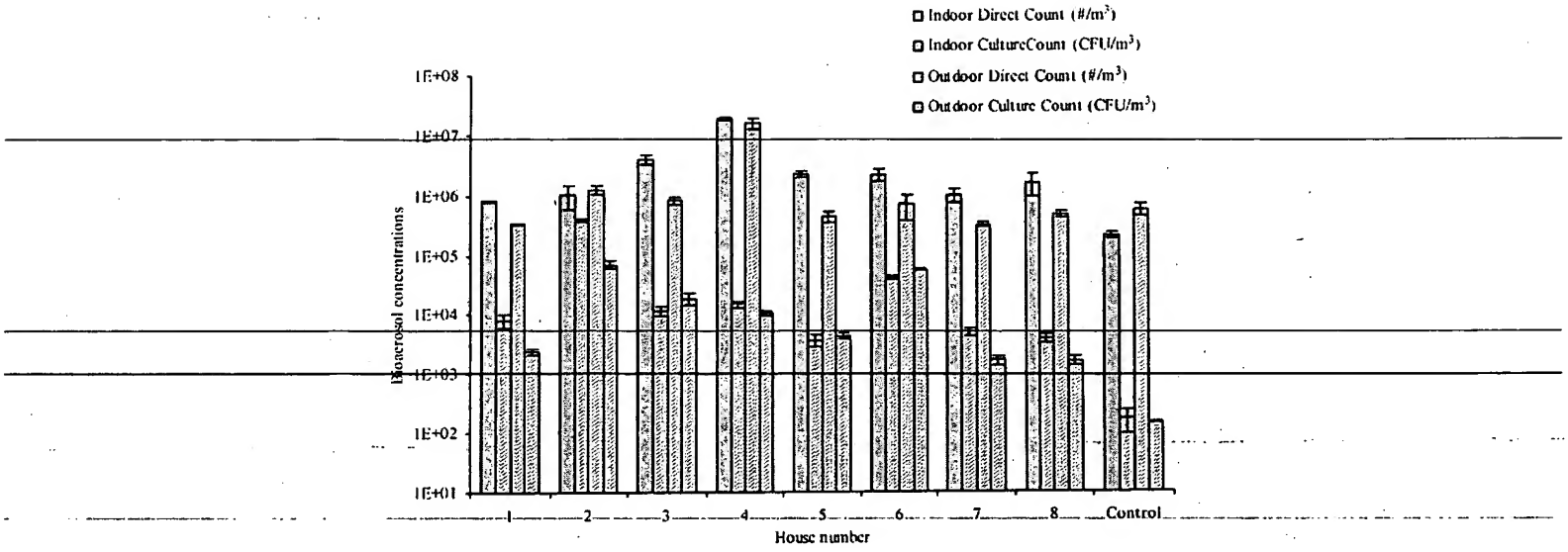


Fig. 6. Direct microscopic counts and culturable CFUs obtained from indoor and outdoor air samples collected with BioSamplers. Error bars represent one standard deviation.

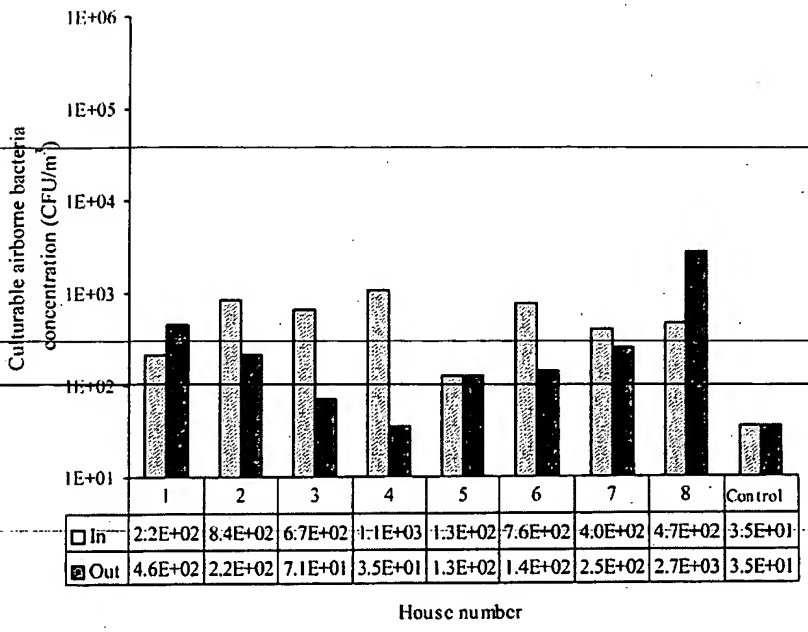
3.5. Comparing direct microscopic counts and culturing recovery from BioSamplers

To compare the recovery of direct microscopic counts and CFUs, both obtained from liquid impinger samples, bacteria and fungi cultured on non-selective media were summed and compared to direct microscopic counts (Fig. 6). Significant differences between concentrations were determined with *t*-tests ($\alpha = 0.05$). Based on direct microscopic counts, seven of eight houses had significantly higher indoor microorganism concentrations compared to outdoors (houses #1, 3, 4–8), a trend which was opposite of that observed in the local control house as well as that reported in larger culture-based surveys (ACGIH, 1999). Based on summed culture counts (i.e. bacteria+fungi), only five houses had significantly higher indoor microorganism concentrations than out (houses # 1, 2, 4, 7, and 8), and no significant difference was observed in the local control house. Indoors, direct counts were 3 to over 1000 times higher than culturable counts obtained from the same indoor air samples while outdoors direct counts were 12 to over 1000 times higher than culturable counts. Although direct microscopy counts were often orders of magnitude higher than culturable counts, these concentrations were poorly correlated (R^2 values 0.004 indoors and 0.02 outdoors). This indicates that culturable counts likely underestimate total microorganism bioburden and cannot predict the magnitude of airborne biological contamination.

3.6. Andersen impactor—culturable recovery

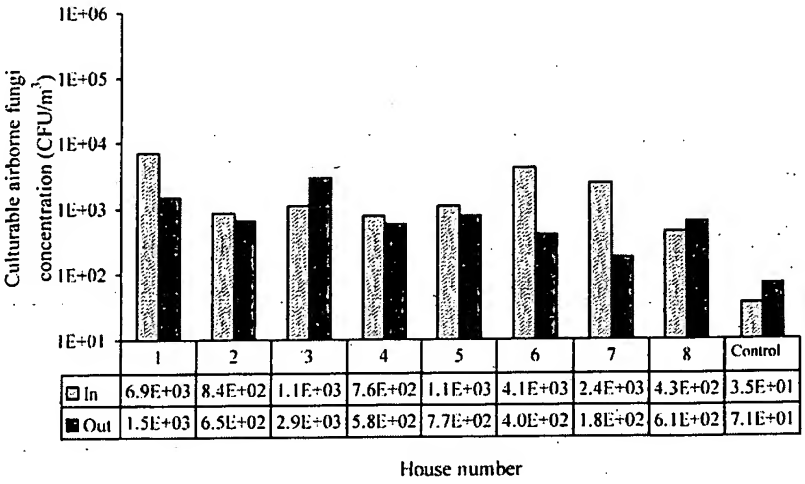
3.6.1. Bacteria

Bacterial colonies cultured on impactor-mounted TSA plates ranged between 1.2×10^2 and 1.1×10^3 CFU/m^3 indoors, and between 3.6×10^1 and 2.7×10^3 CFU/m^3 outdoors (Fig. 7). Inside five out of the eight flooded houses sampled, counts of culturable airborne bacteria were significantly higher (*t*-test, $\alpha = 0.05$) than those measured immediately outdoors, varying between a factor of 1.6 and 30.



	1	2	3	4	5	6	7	8	Control
In	2.2E+02	8.4E+02	6.7E+02	1.1E+03	1.3E+02	7.6E+02	4.0E+02	4.7E+02	3.5E+01
Out	4.6E+02	2.2E+02	7.1E+01	3.5E+01	1.3E+02	1.4E+02	2.5E+02	2.7E+03	3.5E+01

Fig. 7. Estimated airborne concentration of culturable bacteria recovered from one-stage N6 Andersen impactor ($d_{50} = 0.65\ \mu m$).

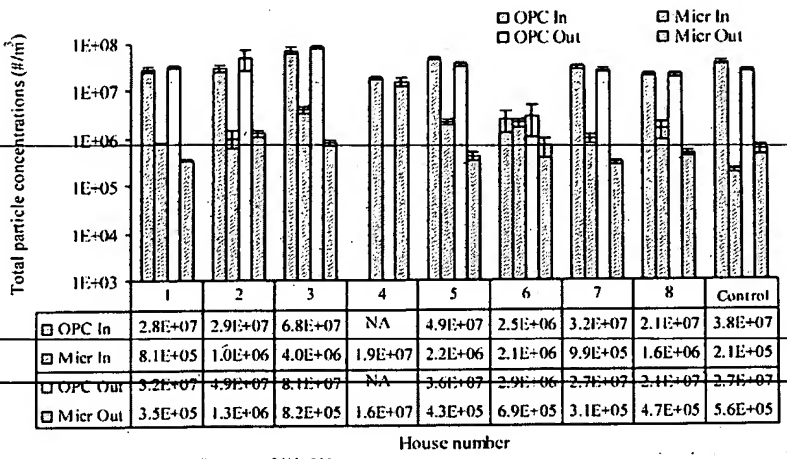


	1	2	3	4	5	6	7	8	Control
In	6.9E+03	8.4E+02	1.1E+03	7.6E+02	1.1E+03	4.1E+03	2.4E+03	4.3E+02	3.5E+01
Out	1.5E+03	6.5E+02	2.9E+03	5.8E+02	7.7E+02	4.0E+02	1.8E+02	6.1E+02	7.1E+01

Fig. 8. Estimated airborne concentration of culturable mesophilic fungi recovered from one-stage N6 Andersen impactor ($d_{50} = 0.65\ \mu m$).

3.6.2. *Fungi*

Concentrations of airborne fungi cultured on MEA plates varied between 4.3×10^2 and 6.9×10^3 CFU/m^{3}indoors, and immediately outdoors they ranged between 1.8×10^2 and 2.9×10^3 CFU/m^3 (Fig. 8). Inside four out of the eight flooded houses sampled, counts of culturable airborne bacteria were



	1	2	3	4	5	6	7	8	Control
OPC In	2.8E+07	2.9E+07	6.8E+07	NA	4.9E+07	2.5E+06	3.2E+07	2.1E+07	3.8E+07
Micr In	8.1E+05	1.0E+06	4.0E+06	1.9E+07	2.2E+06	2.1E+06	9.9E+05	1.6E+06	2.1E+05
OPC Out	3.2E+07	4.9E+07	8.1E+07	NA	3.6E+07	2.9E+06	2.7E+07	2.1E+07	2.7E+07
Micr Out	3.5E+05	1.3E+06	8.2E+05	1.6E+07	4.3E+05	6.9E+05	3.1E+05	4.7E+05	5.6E+05

Fig. 9. Comparison of OPC-measured particle concentrations (OPC) with epifluorescence counts of microbiological particles (Micr), inside and immediately outside flood-damaged and non-flood-damaged houses. Error bars represent 1 standard deviation.

significantly higher (t-test, $\alpha = 0.05$) than those measured immediately outdoors, varying between a factor 1.3 and 13.5.

3.7. *Total particle number concentrations*

Between 70 and 94% of indoor particles, and 62–92% of outdoor particles were measured in the first OPC channel (particle optical diameter between 0.3 and 0.5 μm). Between 4 and 15% of indoor particles, and 5–16% of outdoor particles were measured in the second OPC channel (particle optical diameter between 0.5 and 0.7 μm). Less than 1% of particles observed by the OPC were between 2 and 5 μm. Total airborne particle concentrations indoors varied between 2.5×10^6 and 6.8×10^7 particles/m^3 and outdoors between 2.9×10^6 and 8.1×10^7 particles/m^3 (Fig. 9). Total particle concentration information for house #4 was lost due to equipment malfunction. Indoor and outdoor total particle concentrations were not significantly different in five of the eight flooded houses.

While in all cases, the total particle counts (OPC) were higher than those obtained by direct microscopic counting in corresponding size ranges, the biological contribution to the total particle numbers was markedly different indoors and out. On average, biological particles accounted for 52% of the total particles indoors and 18% of the total particles immediately outdoors, of the flooded houses observed. In the house that did not experience flooding, the trend was reversed, and airborne microbiological particles, respectively, accounted for 3% and 20% of indoor and outdoor airborne total particle numbers. The particle counts from the first channel of the OPC were excluded from this analysis, because whole bacteria and fungi cells typically have diameters greater than 0.5 μm. In order to compare total airborne particle numbers with microbiological particle numbers determined by microscopy, OPC readings from channels counting particles with optical diameters between 0.5 and 5 μm were summed. Particle number concentrations determined by OPC had weak correlation with microorganism numbers collected by the SKC biosamplers ($R^2 = 0.04$ for indoor, $R^2 = 0.14$ for outdoor). Better correlations resulted when OPC

readings for particles with optical diameters $< 0.5\,\mu m$ were included in the comparison: $R^2 = 0.24$ for flooded indoor environments, and $R^2 = 0.18$ for those immediately outdoors.

3.8. Bioaerosol sampling variability and observations of "control" residence

A one-way analysis of variance ($\alpha = 0.05$) applied to microorganism concentrations, both total and culturable, from three impinger sample points indoors showed that the three samples collected at different locations were statistically indistinguishable. The same test applied to the two outdoor sample points yielded the same results.

Total microorganism concentrations in flood-damaged houses were between 1 and 5 times higher indoors than immediately outdoors, indicating an indoor microbial source. For a single non-flooded house included in this survey, the opposite condition existed: the indoor concentration was 33% of the outdoor concentration, which is a value consistent with those commonly observed in non-flood impacted residences and buildings (DeKoster & Thorne, 1995; Lehtonen et al., 1993; Rautiala, Reponen, Nevalainen, Husman, & Kalliokoski, 1998; Robertson, 1997; Yang et al., 1993).

4. Discussion

4.1. Environmental monitoring

Air-exchange rates were monitored concurrently with selected bioaerosols and other airborne particulate matter. The air-exchange rates in the monitored residences varied between 0.8 and 3.5 1/h. This range extends significantly higher than other residential air-exchange rates recorded for the same geographic area and season (Murray & Burmaster, 1995), and may be attributed, at least in part, to the local wind speeds (8.5–16 km/h (daily avg.)). Indoor CO_2 concentrations varied between 300 and 420 ppm in all the houses observed. These relatively low indoor CO_2 concentrations indicated that airborne pollutants are likely not being accumulated because of lack of ventilation (DeKoster & Thorne, 1995).

4.2. Microbial associated volatile organic compounds

The most often observed VOC was 3-methyl-1-butanol, which is a VOC commonly associated with fungal growth. Other VOC measured in flood-damaged homes included: 2-octen-1-ol, 2-heptanone and 1-octen-3-ol. Based on recent literature (ACGIH, 1999; Miller, 1992; Miller, Ross, & Moheb, 1998; Pasanen et al., 1996) the types of VOC measured in the flood-damaged homes were consistent with an indoor enrichment of microorganisms with respect to outdoor sources. Given the relatively high air-exchange rates measured, the levels of specific microbial VOCs were significant in magnitude, and indicate the presence of active generation sources. While some MVOC have been implicated as good indicators of indoor fungal growth, they cannot be used to quantify fungi, either airborne or surface associated, or be related to specific fungi. Nonetheless, MVOC can serve as a signature to the indoor enrichment of environmental fungi given that artificial sources are considered, and that a baseline indoor/outdoor ratio is established. As outlined in review and compared to previous studies (AQS, 1997; Brown, Abramson, & Gray, 1994; Lewis & Zweidinger, 1992), the levels and type of VOC observed in this study were indicative of indoor microorganism enrichment. In the house with the highest MVOC measurement

(House 2) however, a person had smoked prior to air sampling. Tobacco smoke contains hundreds of VOC and some of them may have the same chemical signature as many MVOCs (Molhave, 1992). Five of the eight flooded houses had significant MVOC levels, and these observations corresponded to the houses with the highest averages of culturable airborne bacteria. The house with the highest MVOC concentrations also had the highest culturable microorganism counts recovered from the BioSamplers.

4.3. Comparing culturable airborne microorganism recovery in Andersen impactors and BioSamplers

4.3.1. Bacteria

In five out of eight flood-damaged houses, indoor culturable bacteria concentrations were higher than outdoors (t-test; $\alpha=0.05$). Bacterial CFUs recovered on TSA plates in Andersen impactors agreed with the general trends observed from culturing microorganisms retained in BioSamplers: concentrations of culturable airborne microorganisms recovered from the samples collected indoors were consistently higher than those recovered from outdoors. However, bacteria concentrations recovered with the BioSamplers were significantly higher than those recovered with the Andersen in eight of 9 houses tested, in some cases the differences were greater than two orders of magnitude. A possible reason for these differential recoveries is that the sampling stress incurred by airborne microorganisms recovered by liquid impingers is less than those recovered by impactors. This differential sampling stress response has been previously reported in controlled bioaerosol chamber studies (Stewart et al., 1995).

4.3.2. Fungi

Indoor concentrations of airborne fungi cultured on non-selective medium were significantly higher indoors in six of eight flood-damaged residences.

CFUs from Andersen impactors agreed with general trends observed from culturing fungi from samples retained in the BioSampler: concentrations of culturable airborne fungi recovered from the samples collected indoors were consistently higher than those from outdoor samples. Comparing the concentrations of culturable fungi recovered from Andersen impactors and those retained in BioSamplers, the CFUs recovered by the impactor were between 10^2 and 10^3 times less than those recovered by the impinger. Possible reasons for these differences include: (1) the impinger sampling time (hours), was much longer than the impactor (minutes); (2) retention differences intrinsic to the equipment—impactors are subject to particle bounce where (swirling) liquid capture minimizes particle reentrainment; (3) particle stress—impactor particles are subject to impaction and desiccation, where particles in the impinger were collected in swirling liquid and not subject to impaction and dessication; (4) differences in particle-size collection: the impactor collects particles with a 50% cut-off at an aerodynamic diameter of 0.65 mm, whereas the BioSampler has an efficiency of 79% for 0.3 μm particles, 89% for 0.5 μm particles, 96% for 1 μm particles and 100% for 2 μm particles.

4.4. Epifluorescence microscopic counting vs. traditional culturability assays

In most bioaerosol studies, the detection and quantification of metabolically active microorganisms has been primarily based on plate count assays in which sample collection methods as well as microorganism nutritional requirements and culturability potential bias the results (Hernandez et al., 1999). For this study both culturing and microscopy techniques were used because of the synergy of information that can be obtained from these different counting techniques. Fig. 6 suggests that traditional culturing techniques

are inadequate to represent the true quantities of airborne microorganisms in these indoor environments. Direct counts were 3 to over 1000 times higher than CFUs obtained from indoor airborne particulate matter that was captured in the impingers' reservoirs. Outdoors, direct counts were 12 to over 1000 times higher than CFUs from the same sample aliquots. Even though a high fraction of bacteria and fungal suspended in aerosols may not be viable or culturable, they may retain some potential to induce hypersensitivity and inflammatory disease since such responses have no dependence on microorganism culturability to induce adverse health effects (Flannigan et al., 1991). The investigation adds to a small but growing body of bioaerosol literature suggesting that are formidable differences in culturable and total airborne microorganism numbers present in indoor and outdoor environments. These results suggest that direct counts of airborne microorganisms should be included as a critical component of common exposure assessment paradigms.

Only one home was used in a local control capacity in this study because the literature contains a large bioaerosol monitoring database of non-flood-damaged single and multiple family residences (ACGIH, 1999). These studies report that, under normal residential conditions (no water damage), indoor bioaerosol concentrations are significantly lower than outdoor bioaerosol concentrations during summer season (DeKoster & Thorne, 1995; Lehtonen et al., 1993; Rautiala et al., 1998; Robertson, 1997; Yang et al., 1993). Some of these studies compile observations from over 2000 houses, most of which are based on impactor capture, and independent, broad-spectrum culture of bacteria and fungi as described herein. The results obtained from the "non-flood impacted house" in this study agreed with the large literature database: indoor culturable bioaerosol concentrations were, on average, 33% of outdoor concentrations. With regard to culture-based assays of air samples, this observation is widely reported in the literature not only as the more common residential condition, but the favorable one (ACGIH, 1999).

4.5. Comparison of total particle counts with direct microscopy count

In all cases, the total particle counts (OPC) were higher than those obtained by direct microscopic counting in corresponding size ranges. Differences in microbiological contributions to total airborne particle numbers (both in and outdoors) indicate that this ratio may be a useful index for assessing relative aerosol (bio)burdens in residences flood damaged. As judged by particle numbers, results suggest that indoor sources contributed a significant portion of microorganisms to the airborne particulate matter loads in the flood damaged houses observed. However, weak correlations between direct microscopic counts and total particle counts suggest that optical particle counting will not likely be useful for estimating airborne microorganism concentrations in these environments until a larger data base is compiled.

5. Conclusions

In spite of remediation efforts, indoor bioaerosol concentrations observed in houses with flood water damage were generally higher than outdoor bioaerosol concentrations regardless of the assessment method used. These results are the opposite of bioaerosol concentration trends typically observed in houses with no water damage. Total direct counts recovered more airborne bacteria, fungi and spores than did conventional plate counts. In this study, culturable methods significantly underestimated the quantity of airborne microorganisms both indoors and immediately outdoors of flood-damaged houses—at times this discrepancy was as large as 10^3 microorganisms/m^3.

Commercial air samplers have different collection efficiencies. They can significantly induce sampling stress affecting microbial recovery. The BioSampler consistently recovered a higher fraction of culturable bacteria and fungi than did an N6 Andersen impactor. Given that high efficiency liquid-capture offers capabilities for microscopy concurrent with culturing, and that sampling stress from liquid capture in swirling impingers is significantly lower, BioSamplers offer economical alternatives to impactor-based bioaerosol field studies with added benefits of extended sampling time and control over dilution factors (i.e. no upper detection limit).

The MVOC levels observed in the flood-damaged houses did not correlate with the bacterial and fungal bioaerosol concentrations measured (i.e. the house with the highest bioaerosol concentrations did not have the highest MVOC concentrations). However, given the relatively high air-exchange rates in the residences observed, the presence of MVOC levels indicated an indoor enrichment of microorganisms. While some VOCs are good indicators of microorganism growth, they could not be linked to a specific source or used to quantify the microorganisms from which they originate. The usefulness of MVOC as an index of airborne/surface associated indoor biological contamination may emerge as more studies provide a large enough database to establish VOC correlations to bioaerosol loads observed in the field.

Regardless of source, water can provide significant enrichment potential for microorganism growth on building materials not designed for such exposure, and this enrichment has been implicated to increase indoor bioaerosol levels. There is a lack of studies on bioaerosol exposures following the reoccupation of flood-damaged buildings; previous bioaerosol investigations of these common indoor environments are limited by the conventional culturing techniques used. Drying water-damaged material thoroughly and fast enough to prevent mold or bacterial growth is very difficult, particularly after large-scale water excursions associated with river floods. As part of this demonstration study, all of the houses monitored here were thoroughly cleaned prior to their reoccupation. It is likely that flood-impacted building components, although refurbished, were responsible for the elevated indoor bioaerosol concentrations observed herein. To help evaluate the long-term effectiveness of modern remediation practices, larger, multi-season residential flood surveys of indoor bioaerosol levels should be executed with direct measurements (microscopy, particulate matter and VOC) that provide expanded assessment capabilities complimentary to conventional culturing assays.

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Thermal Treatment: Benefits and Misconceptions of Using High Temperature Heat (>120°F)

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I. Introduction

Man has recognized the importance of heat to cook, dry and sanitize objects since the invention of fire. Within the professional restoration community, higher temperatures are an asset, increasing the ability of air to hold moisture and facilitate drying. There are several successful drying methods available to the restoration professional; a few of the more common are discussed in this paper. Desiccant, refrigerant and convective systems offer practical solutions for the variety of drying situations encountered following water losses and catastrophic events. Though these methods differ in operation and philosophy, they share the underlying principles of lowering the relative humidity and elevating temperature. The temperatures attained by these methods vary depending on the method and the ambient conditions.

Most drying methods generate interior temperatures that range between 90°F and 120°F. When supplemental heat is added to attain temperatures of 120°F to 160°F or more, the objective is to kill bacteria and fungi in addition to drying and desiccating. This "high temperature" procedure is intended to lessen occupant exposure to allergenic components (*i.e.*, mold spores, mycotoxins, fungal mycelia and bacteria) and odors. Some restoration contractors who use high temperature methods refer to their allergen removal process as "pasteurization." Pasteurization is a process to treat bacteria in milk by raising the temperature to 140°F (63°C) for 30 minutes or 161°F (72°C) for 15 seconds. The microbial impacts, health and safety consequences and affects to building construction materials and contents, when employing high temperatures (interior temperatures >120°F) are examined in this paper.

II. High Temperature Effects on Microorganisms and Toxins

Many microorganisms not only survive, but actually thrive at temperatures of 160°F and above. Our ability to state unequivocally that high temperatures kill particular target organisms is predicated on the ability to document sustained, uniform temperatures in the thermal kill zone. Fungi react differently to moist heat and dry heat than bacteria, some may thrive under heat treatment (*e.g., Aspergillus fumigatus* and *Thermoactinomycetes vulgaris*). Some fungi and bacteria are thermophiles, many of which are found indoors. Many fungal spores germinate only *after* heat stimulation treatment and subsequently thrive and grow. Many bacterial spores, particularly Bacillus species (including anthrax) are very resistant to environmental stressors, including heat and can tolerate temperatures higher than that used in high temperature treatment. For example, Anthrax spores can be killed by boiling at 120°C (250°F) or dry heat at 159°C (318°F) for 1 to 2 hours (BioPort

Corporation, 2006). The most extensive studies on the affects of heat on bacteria were conducted in the late 1800's when great scientific interest was created by disease-producing organisms.

Heat resistance has been studied and reviewed historically by several authors (Robertson, 1927; Magoon, *et. al.*, 1926) and produced conclusions that we recognize today. "The subject of heat resistance in microorganisms, in general, recognizes that young cells are more easily destroyed than old cells (Hampil, 1932)." The cause of death of bacteria can be divided into four portions: dry heat and moist heat at both low and at high temperatures (Hampil, 1932.). At low temperatures, dry heat causes the formation of oxidation proteins which destroy bacteria (Paul, Birstein and Reusz, 1910). At high temperatures, two processes may occur. Protein coagulation takes place (Rubner, 1899) and scorching or carbonization of the outside which interferes with nutritive processes.

The complete destruction of bacteria by heat was first studied by Koch (Koch and Loeffler, 1881). These carefully designed experiments formed the fundamental principles of heat sterilization used today. His early experimentation showed that anthrax required a temperature of 284 °F (140°C) for three hours to kill the spores, whereas, they could be killed in a few minutes when boiled. His experimentation demonstrated that steam placed under pressure at elevated temperature sterilized surfaces much more rapidly. Comparisons between the thermal tolerance of different organisms show that complex (animal, protozoa, fungi) organisms will not survive at higher temperatures as compared to simpler organisms (blue-green algae and bacteria) (Table 1).

Table 1. Approximate upper thermal limits for survival in different groups of organisms

Organism	Upper Limit (°F)
Animals including protozoa	113-123 (45-51°C)
Fungi and algae	132-140 (156-60°C)
Blue-green algae	163-176 (73-75°C)
Bacteria	>194 (>90°C)

Source: Brock, T.D. 1967. Life at High Temperatures, Science, Vol. 158, p.1012.

The lethal temperature varies among microorganisms. The time required to kill depends on the number of organisms, the species, pH, duration and temperature (Todar, 2002). Laboratory research shows that the thermal death point for selected pathogenic bacteria and spores range from 131°F and 212°F (Table 2). In a building, the ability to substantiate attaining a lethal temperature is predicated on the completeness of the drying effort, performance measurements and microbial sampling. Even after the living organism is dead, fungal spores, mycelia and mycotoxins still pose an allergenic concern.

High temperature regimes do not destroy mycotoxins (Yang, 2005). The varieties of toxins produced by fungi depend on the species, growth substrate and the presence or absence of competing organisms (Burge and Ammann, 1999). The vast majority of

Table 2. Thermal Death Points of Bacteria and Spores

Species	Duration & Temperature	Author
Well-Known Bacteria		
Bacillus coli	10 min @ 60°C (140°F)	Loeffler, 1886
	30 min@ 65-67°C (153°F)	Escherich and Pfaundler, 1903
	35 min @ 73-75°C (167°F)	De Jong and DeGraef, 1914
	15 min @ 68°C (155°F)	Shippen, 1915
Bacillus thermophilus	5-6 hrs @ 100°C (212°F)	Rabinowitsch, 1895
Bacillus typhosus	10 min @ 56°C (131°F)	Sternberg, 1887
	5 min @ 60°C (140°F)	Bassenge, Mienicke & Friedel, Kolle, Kutscher, 1905
	4 min @ 63°C (146°F)	Orskov, 1926
Paratypoid bacilli	20 min @ 60°C (140°F)	Krumwiede & Noble, 1921
	3 min @ 63°C (145°F)	Orskov, 1926
Dysentery bacilli	1 hr @ 56°C (131°F)	Thomson, 1916
	10 min @ 58-60°C (140°F)	Runge & O'Brien, 1924
Brucelli organisms	10 min @ 57.5°C (136°F)	Eyre, 1912
	5-10 min @ 65°C (149°F)	Zwick & Wedeman, 1913
Bacterium tularense	10 min @ 58°C (133°F)	McCoy, 1912
Hemophilus influenzae	2 min @ 62°C (144°F)	Onorato, 1902
Vibrio cholerae	5 min@ 80°C (176°F)	Koch
	15 min @ 55°C (131°F)	Kitasato, 1889
Bacillus pestis	30min @ 80°C (176°F)	Kitasato, 1894
	2 min @ 60°C (140°F)	Gladin, 1898
	1 hr @ 65°C (149°F)	Kolle, 1912
Staphylococci	10 min @ 62°C (144°F)	Sternberg, 1887.
	35-60 min @ 75°C (167°F)	Samter, 1908
	45 min @ 60°C (140°F)	Neisser, 1921
Meningococci	1min @ 60°C (140°F)	Bettencourt and Franca, 1904
Pneumococci	15 min @ 60°C (140°F)	Wirth, 1916
	30min @ 60°C (140°F)	Bagger, 1926
Pathological Origin		
Streptococci	30 min@ 60°C (140°F)	Ayers & Johnson, 1918
	15 min @ 60°C (140°F)	Wirth, 1926
	24 hr @ 60°C (140°F)	Bagger, 1926
Non-pathogenic Mesophiles		
Str. Lactis	15 min @ 70°C (158°F)	Orla-Jensen, 1919
B. tuberculosis	6 min @ 63°C (145°F)	North and Park, 1917
Clostridium botulinum (Meyer, 1926)		
C. botulinum (Type B)	20min @ 80°C (176°F)	Van Ermengem, 1897
Type A	60 min @ 100°C (212°F)	Thom, Edmondson and Gilener, 1919
Types A and B	240 to 255 min @ 100°C (212°F)	Dickson and co-workers, 1922 & 1925
	300 min @ 100°C (212°F)	Tanner and McCrae, 1923
Type B	10 min @ 100°C (212°F)	Stern, 1926
Anaerobic Spores Clostridium botulinum (Meyer, 1926)		
Cl. welchii	8 - 90 min in steam (>100°C)	Becker, 1920
Cl .nouyi	8 - 90 min in steam(>100°C)	Becker, 1920
Vibrion septique	2 - 15 min in steam(>100°C)	Becker, 1920
Cl.bifermentans	90 - 150 min in steam (>100°C)	Becker, 1920
Other Anaerobic Spores		
Bacillus thermophilus	5-6 hrs @ 100 °C (212°F)	Rabinowitsch, 1895
Bacillus lildzensis	4 min @ 100 °C (212°F)	Karlinski, 1895
Thermophilic Cladothrix	4 min @ 100 °C (212°F)	Kedzoir, 1896
Cl. gelatinosum	75 min @ 100 °C (212°F)	Laxa, 1898
Micrococcus form	10 min @ 76 °C (169°F)	Russel and Hastings, 1902
Bacilla	Restated hours of boiling	Tsilinsky, 1902

Sources: Hampil, Bettylee, 1932. The Influence of Temperature on the Life Processes and Death of Bacteria, The Quarterly Review of Biology, Vol. 7. No. 2, pages 172-196.
Morrison, Lethe E. and Tanner, Fred W. 1924. Studies on Thermophilic Bacteria Botanical Gazette, Vol. 77, No. 2, pages 171-185.

mycotoxins have not been identified; therefore, claims of compete removal following any restoration procedure cannot be substantiated.

III. How Does High Temperatures Work?

High temperature drying requires specific instructions. Recognizing that every project must consider site-specific criteria, we can gain insight into the high temperature procedures in the specifications prepared for the Fresno Housing Authorities (SCS Engineers, 2003). The following is an abbreviated scope of work describing the technical requirements:

1. Purpose

- Drying out moist architectural components.
- Killing viable biological organisms (e.g., insects, fungi and bacteria)
- Oxidizing odors.

2. Site Superintendent:

The contractor will employ a Site Superintendent as the responsible person and will act as an OSHA-Competent Person who can recognize hazards and direct others. The Superintendent is required to record/log all job-site work progress. The Superintendent shall be fully qualified through education, training and experience to perform the work.

3. General Pre-start Inspection

The Superintendent must perform an inspection of the structure before assembling heat generators or distribution equipment. The inspection will verify if the structure is safe and sound and will not be compromised when heated, and whether the structure is devoid of personal belongings. These observations will be recorded.

4. Site Set-up

The Superintendent shall layout the heat generators and distribution equipment to ensure that the heat can be equally distributed within the designated area. The layout of the heating equipment shall be recorded on a drawing. All salient features of the structure shall be recorded on a drawing. The Superintendent shall ensure that the heat generators are sufficiently sized to bring the structure up to specified target temperatures and maintain those temperatures for the specified duration.

5. Establishing Temperature Monitoring Points

To be effective, a threshold temperature must be maintained for a specified duration of time. Temperatures must be measured and recorded during the entire heat treatment process. Temperature must be measured in real time in the air space and within various architectural components.

6. Threshold Temperature

The threshold temperature shall meet or exceed 160°F or 71°C in the majority of probes. All probes in the designated heat treatment area shall reach a minimum temperature of 155°F or 68°C. However, temperatures in the structure shall not exceed 175°F or 80°C.

7. Temperature Duration

The duration with which most temperature probes shall be maintained above the threshold temperature (160°F) is 60 minutes. All probes in the designated treatment area shall be maintained at or above the threshold temperature for a minimum of 60 minutes.

8. Cool-down Period

Upon meeting temperature and duration goals, a 60-minute cool-down period shall be initiated. During the cool-down period, all heat sources are turned off and the structure is to remain sealed while temperature monitoring continues.

Insight provided by high temperature drying practitioners offer practical perspectives. "You can't achieve high temperatures until the wood is dry" (Vyrosek, 2006). Moisture content can vary widely in a flooded home where some wood members may be saturated while other materials may contain sufficient water content to support microbial growth (approximately 20% or greater moisture). Both types of materials, saturated or elevated moisture, can be dried at temperatures less than 120°F. Proper drying, however, must consider the wide range of moisture content.

The duration of drying is critical and should be carried out slowly and uniformly, typically over a two, three or four-day period, depending on the circumstances. As in kiln drying (see below), differential temperatures and rapid temperature changes increase the possibility of damage as wood dries. Crawl spaces with exposed soil, heat sinks (i.e., concrete and brick structures) and building envelope breaches pose restrictions to achieving temperature uniformity.

Is High Temperature Necessary?

This is perhaps the most important question of all. The answer depends on whether high temperature is the most practical, efficient and safe method to meet the client's needs. Sewage-flooded structures, schools and medical facilities compromised by pathogenic microorganisms, insect-infestations, nuisance odors and individuals with multiple chemical sensitivities may benefit from the high heat process. However, the public's understanding of a "unique" or "innovative" restoration strategy may be clouded by their perception that it is also the "most appropriate."

The public's perception of mold is influenced by their health, personal observations and the media. When water damage and resulting microbial growth occur, some consumers seek comfort in absolute remedies, "I don't want a single *Stachybotrys* spore in my house," or "the indoor air quality should pose no health risks from mold." Though satisfying these expectations are always short-lived, if not impossible to achieve, business is sometimes driven to satisfy the loftiest expectations. In some circumstances the risks associated with remediation may exceed the benefits.

A recent indoor air quality article examined whether microbial growth can be left inside walls based on the relative risk (Burge, 2005). Some fungal species (*e.g. Penicillium chrysogenum*), though prevalent after a water loss, pose no risk when enclosed in the wall cavity of a school. Though inconsistent with parent and teacher expectations, the practical aspects of removal (*i.e.*, school closings, restoration costs, loss of salary and expenses) offer a sobering perspective of mold's presence and its priority for removal. The most appropriate remediation technology should be elected wisely by considering the inherent risks.

How Do We Know It Worked?

Performance measurements collected before, during and after any restoration procedure are necessary to confirm that the restoration process met the design intent or intended outcome. Fungi and bacteria are present on the entire earth's surface; determinations of effective thermal kill and removal must be documented in periods measured in minutes and hours after project completion. Once the structure returns to ambient temperature, moisture will return the construction materials and contents to their equilibrium moisture content; ventilation and infiltration will inoculate a host of fungal and bacterial species into the interior spaces within a few days.

In the end, the most effective way to reduce future microbial proliferation is to keep the interior dry. A detailed logbook describing the moisture content at multiple sampling locations, temperature and relative humidity measurements, microbial sampling of air and surfaces and infrared analysis of the structure will support a successful project and substantiate payment.

Elevated temperatures not only desiccate microbes, they also accelerate aging and change the performance of various materials. The potential effects on building materials are described below.

IV. High Temperature Affects on Building Materials

Wood

The behavior of drying wood in commercial kiln operations underscores the importance of slow and consistent drying effort at high temperatures. Structural wood, exposed to elevated temperatures, experiences the same potential damage as wood in a kiln (Simpson, 1983).

Water is present in three (3) forms in wood cells; 1) liquid or free water, 2) water vapor, and 3) chemically bound or hygroscopic water. When wood dries, free (cellular) water is the first to leave the cell. When the cell is dry, the cell walls still contain chemically bound (hydroscopic) water. This stage of drying is termed the fiber saturation point (fsp). Wood cells will not shrink (distort) until bound water is extracted from the cell wall. The fsp is a critical milestone in the drying process because wood strength and shape will not change until the moisture content falls below the fsp.

When lumber is dried too quickly, drying stresses and damage can affect the strength of structural members in the home (USDA, 1957). Damage is caused by two kinds of stress, hydrostatic tension and differential shrinkage. Hydrostatic tension is created where high drying temperature build up the hydrostatic tension in a cell. As a result, the interior cells collapse and there is an appearance of excessive shrinkage and a washboard effect in lumber.

Differential shrinkage occurs between the shell and center of the lumber when the outer wood fibers dry and shrink before the inner wood cells have begun to dry and shrink. This condition is termed, "case hardening." When this occurs, the core moisture cannot pass through to the surface. This prevents proper "wicking" to the surface of the board and evaporating. Lumber that is dried too quickly will degrade during the initial stages and will slow the overall drying process.

Wood dries the fastest at the beginning because the moisture differential is at its highest. This is when wood and a home are most susceptible to damage. During the early stages of drying, low temperatures and high humidity are necessary for many species of wood. As drying progresses, the temperature is slowly raised and the humidity lowered to maintain a steady drying rate

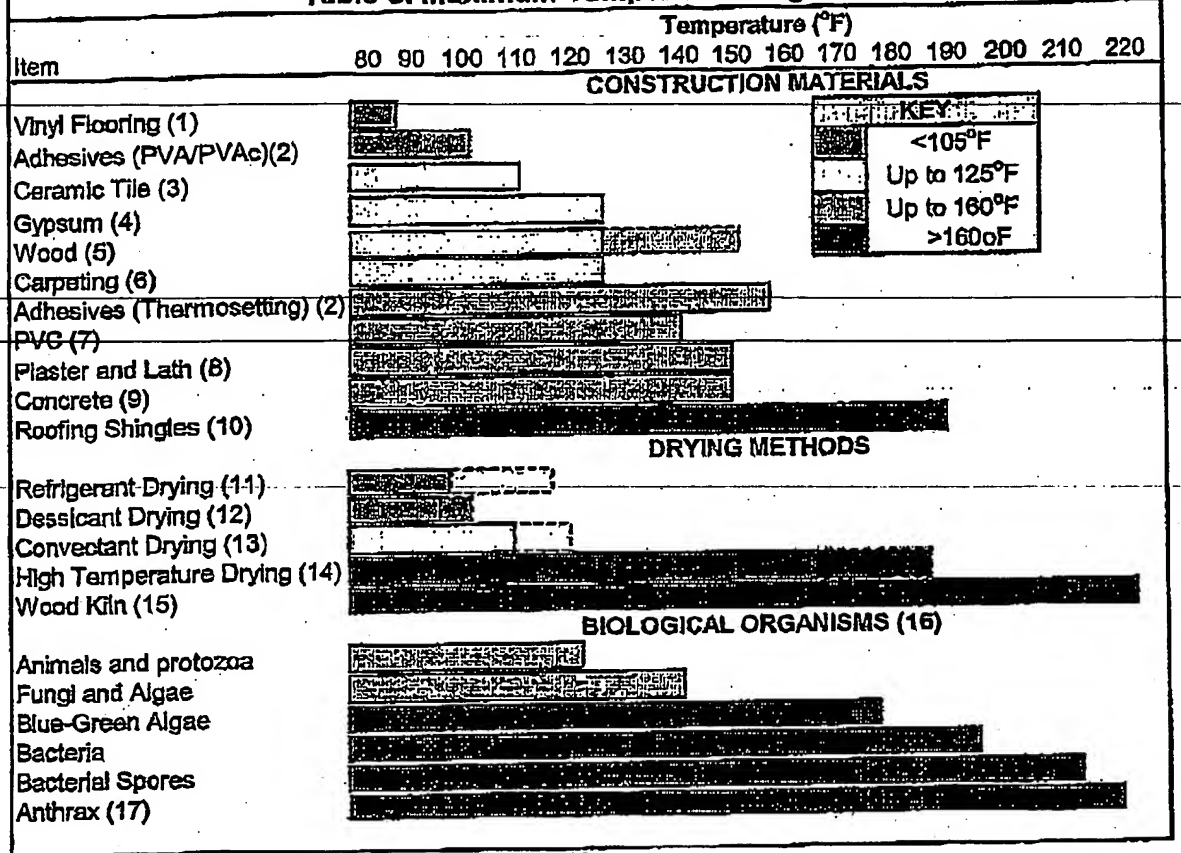
Wood: High Temperature Research

Research performed on wood roofing systems (beams and sheathing) performed well when exposed to temperatures up to 150°F (Table 3). Roof systems experience elevated temperatures *via* solar radiation [American Forest and Paper Association (AFPA), Inc., 1999].

Temperature measurements of roof systems vary depending on the orientation, hour of day, season, color, ventilation rate and insulation thickness. Seasonal measurements showed that roof systems reached 150°F for short durations; the hottest members were limited to roof sheathing.

Under the severest conditions, the temperature of the structural beams, rafters and truss members in wood roofs generally do not reach 140°F. However, when these conditions do occur, the loss of strength associated with increased temperature is compensated by the increase in strength associated with lower moisture content.

Table 3. Maximum Temperature Regimes



1 Resilient Floor Coverings Institute, Freeman, 2006
2 Conner, 2001.
3 Lafortune, 2006.
4 Gypsum Association, 2004.
5 American Wood Council, 2005
6 Mohawk Industries (unofficial)
7 Harvel Engineering, 2006
8 New York Plaster and Lath Institute, Bill Hohlfield, 2006.
9 American,Concrete Institute, Tholen,M. 2006.
10 GAF Materials Corporation, 2006 Technical Services
11 DRI EAZ LGR 2000, Phoenix 200 HT will operate as high as 120oF.
12 Mellcon Industries
13 Cressy, 2006 (personal communication)
14 Hedman, 2006 (Personal communication)
15 Simpson, 1983-84, Drying Technology
16 Brock, T. Life at High Temperatures, Science, Vol. 158, p. 1012
17 BioPort Corporation, Lansing Michigan (1-877-246-8472)

Research results conducted during short-term, high temperature exposures has shown an increase in wood strength properties when cooled below normal temperatures and a decrease in properties when heated above 150°F (AWC, 1991). When the wood returned to a normal temperature, it recovered its original properties.

Research found that examined wood exposure at temperatures above 150°F showed a permanent loss in strength when cooled and tested at normal temperatures. These permanent effects were additive to those that occurred at the exposure temperature. Permanent strength losses occurred following exposure to temperatures >212°F; the damage was greater when wood was heated in water rather than in dry air.

Based on this research, temperatures of 150°F represent a threshold for the beginning of permanent loss of strength. This interpretation was substantiated by test data that showed an approximate 10% loss in bending strength for materials exposed for 300 days in water at 150°F and then tested at room temperature. The use of lumber or glued-laminated timber members that experience long-term exposure to temperatures over 150°F, should be avoided as shown below.

Heating Duration	AWC DWS Design Specifications
Short term heating up to 150°F	No design strength reduction required
Sustained temperatures ,100°F	No design strength reduction required
Sustained temperatures 100°F to 125°F	10% to 30% design strength reduction, depending on the moisture content.
Sustained temperatures 125°F to 150°F	10% to 50% design strength reduction, depending on the moisture content and specific property.

Source: AWC, 1991.

Wood: Adhesives

The vulnerability of adhesives to elevated temperature regimes (130-160°F) depends on the chemical structure of the adhesive used (Conner, 2001). Wood adhesives are generally classified as either synthetic or natural **(Table 4)**. Synthetic adhesives are derived from petroleum products. These adhesives are usually applied as a water-soluble liquid to the wood surface. Adhesive prepolymers cure by reacting further to form polymers at the contact point. Heat and cross-linking chemicals are often added to strengthen the curing reactions.

Synthetic adhesives are classified further as either thermoplastic or thermosetting resins. Thermoplastic resins such as Polyvinyl acetate (PVAc) ($CH_3COOCH{=}CH_2$) and Polyvinyl alcohol (PVA) ($-CH_2-CH(OH)-_{(n)}$) soften when exposed to heat and solidify when cooled to room temperature.

Thermoplastics are more vulnerable to elevated heat. PVAc is most widely used as an emulsion that is white to off-white in color that is used in many household applications. Commercial uses include laminating adhesives, floor tiling and paper coatings. When

Table 4. Wood Adhesives Comparison

Class	Nature	Resin Type	Adhesion System	Form and Cure	Strength Properties	Uses	Vulnerability (1)
Synthetic	Thermosetting	Amino	Urea-Formaldehyde (UF)	Powder and liquid forms. White to tan with colorless borderline	Durable under damp conditions, low resistance above 122°F	Hardwood plywood furniture, fiberboard Underlayment, flush doors	HIGH
			Melamine-formaldehyde (MF)	Powder with blended catalyst; up to 40% urea. White to tan	High dry and wet s strength. Resists water and dampness	Hardwood plywood and jointing, edge-gluing	LOW
			Melamine-Urea-formaldehyde (MUF)				LOW
		Phenolic	Phenol-Formaldehyde (PF) Resorcinol-formaldehyde (RF) Phenol-resourcinsol-formaldehyde (PRF)	Liquid, powder, and dry film, dark red bondline Cured hot (>120°F)	High dry and wet strength. Resists water and dampness	Primary adhesive for exterior softwood plywood	LOW
		Isocyanate	Diphenylmethene- 4,4'-diisocoyanate (MDI	Liquid emulsion and separate isocyanate White with hardener colorless borderline	High dry and wet strength. Resists water and damp atmospheres.	Laminated beams laminated plywood to steel and plastics	LOW to MOD
		Epoxy	Bisphenol A-based epoxy resins	Liquid resin and hardener	High dry and wet strength. Resists water and damp atmospheres	Laminated veneer and wood boat hulls aircraft components wood beams and railings	LOW
		Elastomeric	Styrene butadiene rubber (SBR)	Putty-like in consistency Tan, yellow or grey	Strength develops slowly. Resistant to waer and moist atmospheres	Decorative kitchen countertops wood and paper lamination	LOW to MOD
	Thermoplastic	Vinyl	Polyvinyl acetate (PVAc) Polyvinyl Alcohol (PVA)	Liquid applied ready to use. Dries off white.	High dry strength. low resistance to moisture and elevated temperatures	Interior and exterior doors and moldings Architectural woodwork Furniture	HIGH HIGH
		Hot Melts	Ethylene vinyl acetate (EVA) Polyurethane	Solid blocks, pellets ribbons, rods, or films White to tan; near colorless bondline	Strength develops quickly on cooling. Moderate resistance to moisture	Edge-banding of plastic plastic lamination paper overlays furniture assembly	LOW LOW
Natural		Protein	Casein	Powder with added chemicals, white to tan borderline	High dry strength Moderate resistance to water and dampness	Interior doors laminated lumber	MODERATE
			Soybean	Powder with added chemicals. White to tan. Same borderline	Moderate to low dry strength. Low resistance to water and dampness. Moderate resistance to intermediate temps.	softwood plywood fingerjoints for lumber	MODERATE
			Blood	Solid and partially dried whole blood. Dark red to black bondline	High dry strength: Moderate resistance to water and dampness and organisms.	Interior doors discontinued use in laminated lumber	MODERATE

References: Forest Product Laboratory, Wood Adhesives, Science and Technology FS-FPL-4703
A.H. Conner, 2001

(1) Vulnerability is generally considered temperatures between 130 and 180°F.

exposed to elevated temperatures (100°F), PVAc will soften and become less resistant to high moisture and humidity than thermosetting resins.

Thermosetting adhesives (*i.e.*, amino resins, phenolic resins, epoxy resins and isocyanates) are the principal type of adhesive used to bond wood and are less vulnerable to heat. The principal difference between thermoplastics and thermosetting adhesives is that thermosetting adhesives form polymers that cross-link when they cure. When cross linkage occurs, the cured adhesive is insoluable and does not soften when heated.

Natural adhesives are derived from starch, soybean, animal waste and meat processing and tanning by-products and casein from skim milk. Protein based adhesives (*i.e.*, soy, blood and casein) are the most common; however, these adhesives are most often used for interior applications. Natural adhesives are used as a water-soluble application and cure when the solvent (water) is removed. Some formulations add chemicals to aid in cross-linking to enhance strength. These additives lessen the vulnerability of natural adhesives to high temperatures (130 to 160°F). The primary disadvantage of natural (proteinaceous) as compared to synthetic adhesives is their vulnerability to microbial degradation and lower resistance to moisture.

High Temperature Affects on Building Materials

A summary of maximum temperatures recommended by selected construction material institutes and building material manufacturers is summarized in **Table 3.**

Gypsum

Gypsum should not be exposed to temperatures above 125°F for extended periods. The Gypsum Association (GA) provides written specifications for the "application and Finishing of Gypsum Panel Products) (Gypsum Association, 2004). GA technical documents state the following recommendations:

1.4 Gypsum panel products shall not be used where they will be exposed to sustained temperatures for more than 125°F (52°C) for extended periods of time.

1.5 Where gypsum panel products are used in air handling systems, the surface temperature of the gypsum panel products shall be maintained above the air stream dew point temperature but not more than 125°F (52°C).

Plaster and Lath

Technical representatives of the New York Plaster and Lath Institute expressed confidence that temperatures between 120°F and 160°F would have no effect on the integrity of a plaster and lath wall (Hohlfield, 2006). The only circumstances that might

pose an exception would be a recently constructed plaster wall that had not yet cured. Plaster curing requires approximately 30 days.

Roofing Shingles

Asphalt shingles are durable at high temperatures because the minimum softening point is approximately 190°F (GAF, 2006). GAF technical staff expressed three concerns. The adhesive sealant used in the GAF shingle is "Dura-Grip." This adhesive has a lower softening point than the shingle and may "ooze" at lower temperatures. Should this occur, it will become evident immediately after the heating event. After exposure to elevated heat, shingles are prone to "slippage," as a result, it is not recommended to access the roof until the shingles reach ambient temperatures. Finally, elevated temperature may accelerate the aging process in the shingles. The effects of aging may not be visible.

Concrete

Concrete is generally resistant to damage when heated between 120°F and 160°F (Zalesiak, C. 2001). The literature is sparse on the effects of heat on concrete at high heat restoration temperatures; however, detrimental effects may occur depending on the rate of heating (Tolen, 2006). Very rapid concrete surface heating will result in rapid expansion and cracking. The most relevant guidelines were offered by the American Concrete Institute (ACI) and were described in ACI 349R-01 Appendix A, "Code Requirements for Nuclear Safety Related Concrete Structures."

> A.4.1 - The following temperature limitations are for normal operation or any other long-term period. The temperatures shall not exceed 150°F except for local areas, such as around penetrations, which are allowed to have increased temperatures not to exceed 200°F.

Vinyl Flooring

Vinyl floor coverings are tested by the Resilient Flooring Institute (RFI) using ASTM F1514, heat stability tests (Freeman, 2006). The test elevates the temperature of the flooring to 158°F for seven (7) days. After this period, the flooring exhibits noticeable discoloration. The maximum temperature recommended by the RFI is 85°F; this temperature is based on radiant heat temperatures achieved during the heating season. The warrantee for resilient flooring that exhibited discoloration after heating may not be honored by the manufacturer.

Ceramic Tile

Large expanses of ceramic floor tiling ranging from 30 to 60 feet in length would pose a problem at temperatures ranging between 130°F and 160°F (LaFortune, 2006). Floor tiles vary in their thermal expansion coefficients depending on their composition;

therefore, a specific statement on their performance at these temperatures cannot be made. Most floor tile installers do not strictly follow industry guidelines for the construction of expansion joints. As a result, any forgiveness in the ceramic tile flooring at elevated temperatures may not be provided by expansion joints. Based on field reports for radiant floor heating systems, maximum temperatures that range between 100°F and 110°F have not shown problems. Temperatures that exceed 160°F are likely to experience tile release from the floor.

PVC Products

Polyvinylchloride (PVC) is used in an extensive array of products including electrical components, wiring insulation and coatings, membranes, water supply piping, exterior windows and window shades, home appliances, tables and chairs. The maximum service temperature is 140°F with heat deflection at 170°F (Harvel, 2006).

V. Safety Concerns

The use of temperature regimes above 120°F poses a higher level of care, training and OSHA and USEPA scrutiny than any other restoration strategy. Four areas of concern (*i.e.*, direct-fired propane heaters, dust, heat stress and property damage) are described below.

Direct-Fired Propane Heaters

When used properly, propane is an odorless and colorless gas that is safe and convenient (OPA, 2006). Propane leaks, however, pose an immediate hazard because propane settles in low spaces and a low concentration can create a flammable mixture. In a confined space, a propane gas leak poses an explosive hazard.

Propane requires a large volume of air to burn correctly; one cubic foot of propane requires 23.5^3 ft. of air. The proper mixture of air and fuel is essential because too much fuel will result in incomplete combustion and the formation of carbon monoxide.

Carbon Monoxide poisoning occurs when, carbon monoxide preferentially attaches to the blood molecule (hemoglobin) that carries this gas instead of oxygen. A person with carbon monoxide poisoning is overcome by carbon monoxide (instead of oxygen) and immediately feels light-headed, dizziness and/or nausea (DHHS, 2005). Prolonged exposure may result in death. When direct-fired propane burning heaters are used, monitoring of carbon monoxide and proper ventilation with fresh air are required for safe operation and the protection of personnel.

Dust

Turbulent fans assist the drying process; however, they also aerosolize microbial matter and dust. As a result, turbulence also creates potential combustible conditions by the emancipated dust. A cloud of dust, within its flammable concentration limits, will not

burn unless sufficient energy is provided to ignite it such as open flames (*i.e.*, propane heaters) and hot surfaces (dryers, heaters, etc.) (DSEAR, 2002). Both of these conditions are present with the use of a direct-fired propane heater. Safety information provided by the manufacturer of propane forced air heaters reinforces these concerns (DESA, 2006). This safety hazard can be reduced with indirect fired heaters.

Heat Stress

Drying and heating processes that involve high temperatures, radiant heat sources and high humidity can induce heat stress. The human body maintains a fairly constant temperature even though it is exposed to a range of temperatures (U.S. DHHS, 1986). As the surrounding temperature approaches the skin temperature, cooling becomes more difficult. Increased body temperature and physical discomfort promote irritability, anger and other emotional conditions that may prompt workers to overlook safety procedures and divert attention from hazards.

The current permissible heat exposure threshold limit values (TLVs) pose limitations for light work at 90°F for 15 minutes of work and 45 minutes of rest 9ACGIH 1992). The ACGIH TLVs state that,

> Higher heat exposure than those in **Table 5** are permissible if the workers have been undergoing medical surveillance and it has been established that they are more tolerant to heat than the average worker. Workers should not be permitted to continue work when their deep body temperature exceeds 38°C (100.4°F).

Table 5. Permissible Heat Exposure Threshold Limit Values

Work/Rest Regime		Work Load		
Work	Rest	Light	Moderate	Heavy
100%	0%	86°F	80°F	77°F
75%	25%	87°F	82°F	78°F
50%	50%	89°F	85°F	82°F
25%	75%	90°F	88°F	86°F

Source: U.S. Dept. of Labor, 2006. (The table number changed for this paper.)

The OSHA Technical Manual states, "Every worker who works in extraordinary conditions that increase the risk of heat stress should be personally monitored. Personal monitoring can be done by checking the heart rate, recovery rate, oral temperature, or extent of body water loss."

Property Damage

High temperatures (>120°F) increase the likelihood of property damage (**Table 3**). PVC -containing products, thermoplastic adhesives, wood, carpeting, and vinyl flooring

represent products and materials that may be damaged irreversibly. Furthermore, manufacturer recommendations for the maximum exposure temperature may cancel out product warrantees.

VI. Conclusions

High temperature or "Pasteurization" restoration techniques pose both creative restoration opportunities and elevated risks. Structures that are contaminated with pathogens or support extensive microbial contaminants may benefit from desiccation and the capture of microbial mass.

Varying degrees of property damage are inevitable with this restoration procedure. Efforts to remove and protect contents are essential for customer satisfaction and to lessen potential liability.

Heating beyond 120°F requires an exceptional level of safety training, personal protection and a detailed understanding of combustible and explosive environments. Unforeseen safety hazards and accidents will undoubtedly initiate OSHA's scrutiny in the workplace. High temperature procedures will place safety as an extreme concern on the jobsite.

Client expectations are bound to soar if "high temperature restoration" is marketed as a sanitation technique. Historical studies on thermal death in bacteria and spores ranges as high as 212°F, making sanitation and unachievable goal.

No restoration process is permanent. High temperature is clearly a benefit in the short-term; however, structures where the moisture content is poorly regulated will return to their previous condition. Client training and orientation to the importance of maintaining building performance and moisture controls may help lessen claims of misrepresentation.

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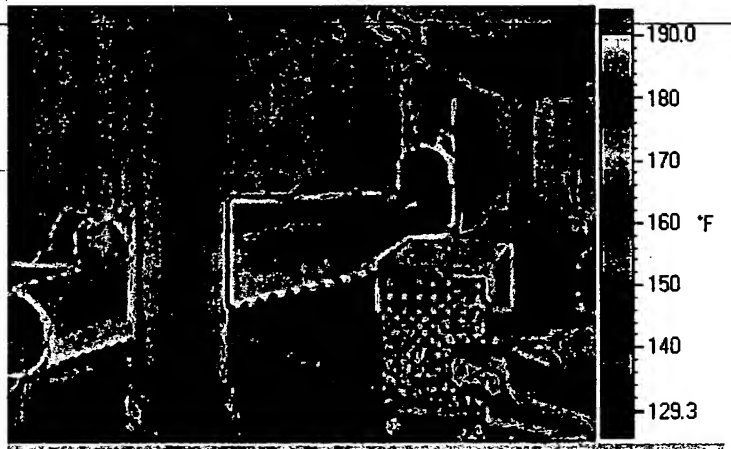
Heat Treatment Method Provides Water Damage/Mold Relief

Escalating water damage and mold liability could cost insurers and property owners

By Alan Forbess

For insurers facing extraordinary exposure from Hurricanes Katrina and Rita, the bad news continues. Serious mold contamination is now threatening water-damaged homes and commercial properties throughout the region. With losses estimated to rise beyond $90 billion in flooded New Orleans and the Gulf Coast, the more than 15,000 adjusters dispatched aren't nearly enough to handle the region's estimated two million claims. Hurricane Wilma and recent flooding in the Northeast are only compounding the problem, for where there's flooding that is not addressed immediately, mold growth and resulting claims will surely follow. Moreover, the hurricanes may just be the start of painful times for insurers and property owners if the disruptive weather patterns predicted for the rest of the century by Purdue University researchers prove correct.

With all this bad news piling up, the insurance and real estate industries could use some good news for a change. A revolutionary new heat treatment process established in California is looking like it could be the silver lining to a very cloudy period, providing an alternative methodology which could save the insurance and real estate industries billions of dollars. ThermaPureHeat may be a big part of the solution. ThermaPureHeat has proven to be an effective alternative to traditional demolition-based remediation and building dry-out methods, potentially saving US insurers billions of dollars over the next several years.



Heat has shown to be effective in destroying active mold growth sites, and kills viable mold spores, bacteria, viruses, insects, and other heat-sensitive pests and organisms.

The process, developed by E-Therm, an environmental remediation innovator based in Ventura, Calif., uses superheated, dehumidified air to disinfect, decontaminate, and dry out buildings in much the same way heat is used to pasteurize milk and kill bacteria in wine.

In the ThermaPureHeat process, technicians use propane-powered portable heaters and air blowers to inject superheated air into the affected space, raising the temperature of a single room or entire structure to as much as 160 degrees Fahrenheit for several hours. Heat has shown to be effective in destroying active mold growth sites, and kills viable mold spores, bacteria, viruses, insects, and other heat-sensitive pests and organisms. Heat also accelerates the off-gassing of odors and toxins, even in inaccessible areas, without the use of chemicals. One of the main benefits of heat is that the proper application can dry out wet buildings much quicker than the traditional method of simple air movement and dehumidification typically used by flood restoration contractors.

Whether applied to aid in disaster recovery or in addressing

more routine water intrusion problems, insurers and property owners are finding that heat offers an effective alternative or adjunct to costly traditional demolition-based mold remediation and flood restoration.

Used in conjunction with limited "remove and replace" remediation or as an alternative to it in some instances, the heat treatment process could minimize liability and increase clearance testing success rates. Heat also allows the contractor to treat many building materials in place, avoiding the cost and expense of unnecessary removal of walls, flooring, cabinetry and furnishings.

Drawbacks of Traditional Mold Remediation

Traditional mold remediation typically includes limited or extensive demolition of impacted building materials, and extensive cleaning using techniques such as wire brushing, sanding, HEPA vacuuming and microbial wipe down. This has been the standard mold remedy, which is costly and time consuming. As with all response actions, the more extensive the tear down the higher the build back costs.

"Cost escalates when suspected mold requires the tear down and build back of structures that may be salvageable," says Joe McLean, CEO of Alliance, a Calif.-based environmental contractor that deals extensively in mold and asbestos remediation. "For instance, if a consultant specifies removal of a 4-foot perimeter on four walls because moisture has wicked up one, the tear down and build back of showers, cabinets, countertops and such can significantly increase costs."

Because insurers often cover building structures, their contents, as well as loss of use, long remediation projects that vacate the occupants for weeks or months can also rack up high secondary costs. The cost for replacement housing, meals - or more significantly, the cost of insuring lost business - can, in fact, sometimes exceed remediation costs.

Inaccessible areas such as wall cavities, crawlspaces, headers, doorjambs, and vapor barriers present another dilemma. Either spend prohibitively to reach, remove, and replace building structures in these inaccessible areas - or leave them with potential live mold or mold spores which could pose a re-infestation hazard.

Removal and replacement of mold-affected areas can also be complicated by other factors - such as when building structures like studs or floor joists are structurally necessary, or when historical features such as frescos, carved wood, or decorative plasters prove difficult or prohibitively expensive to replace.

Reining in Mold Liability

Some in the industrial hygiene community feel that the sky-high cost of mold liability can be brought back down to earth by refocusing on the basics.

"Mold remediation today is stuck in the mindset of early asbestos remediators who believed that everything had to be ripped out regardless of the cost," says Michael Geyer, P.E., C.I.H., C.S.P., who's President of Kerntec Industries, a Calif.-based environmental consulting firm. "Remediators later learned that asbestos could be more effectively managed in place at lower cost in many instances; the same is true of mold today."

According to Geyer, the industrial hygiene community has been focusing on the symptom - mold - while failing to properly address the cause - moisture.

"If physical removal is the only acceptable remediation method, you may as well demolish the building," says Geyer. "Because you can't simply scrub mold off the surface when its roots grow into the substrate." Geyer explains that mold, as a fungus, is a plant without chlorophyll whose roots grow into the substrate of building materials and whose spores are like the seed-bearing fruit of a tree.

"To properly handle mold, you have to handle the moisture problem," adds Geyer. "Applying heat through a process like ThermaPure's is not only lethal to mold and other biohazards like bacteria and insects, but it also dries out the substrate, structure, and architectural elements. This helps prevent future recurrences since the substrate is no longer hospitable to growth."

"Mold in a wall cavity doesn't necessarily need to be removed as long as it's effectively killed and not part of the occupied space," says Geyer. "In instances of mild to moderate water intrusion of short duration, substrate removal is usually unnecessary and unwarranted except when visibly

ThermaPure Heat also accelerates the off-gassing of odors and toxins, even in inaccessible areas.

contaminated or when architectural elements are compromised. That's where heat treatments like ThermaPure can be effective for managing mold in place. It penetrates cracks, crevices, and typically inaccessible areas like wall cavities at a fraction of the cost of removal and replacement."

Don't Demolish the Bottom Line

When a water loss incident with detectable but no visible mold affected office space at a Juvenile Hall in a Monterey County, Calif., gross removal including the impacted wall cavity was estimated at $20,000. Instead, the County opted to manage the mold in place using the ThermaPure process. The impacted area was heated to 160 degrees Fahrenheit while maintaining 145 degrees Fahrenheit in wall cavities and other inaccessible spaces in excess of two hours. Mold remediation protocol including critical barriers, negative air containment, and HEPA vacuuming were implemented as well.

Afterward, post remediation viable samples analyzed by Hygeia Labs of Pasadena, CA revealed no viable mold/fungi detected within the impacted wall cavity. Costly gross remediation was avoided and inaccessible areas received additional drying. The savings to the County using ThermaPure in lieu of gross remediation was $17,000.

Because ThermaPure treating a structure generally takes less than eight hours, no multiple day move outs are required. This minimizes business disruption and loss as well as any secondary costs such as for housing or meals.

A Case Study

Recently, a large investment group purchased a student housing complex at a major Southern California university. During the due diligence period, building inspections revealed water damage or elevated moisture levels in 109 of 122 residential units, along with an extensive termite problem. Complications included an accelerated restoration schedule, budget constraints, and a summer occupancy schedule which was already booked.

The consultant recommended the ThermaPureHeat process to restrict demolition to only those areas where physical damage or visible mold growth was present. Of the 109 units needing remediation, only 10 units required extensive demolition, including cabinetry or shower stall removal. ThermaPure effectively killed the mold in inaccessible areas, allowing minimal removal and replacement as part of site remediation.

This significantly cut required restoration time and costs. All units were HEPA cleaned and sampled as part of traditional post remediation testing, with all 122 units passing. By working in selected buildings and moving quickly through the complex, the university was able to house specialty groups and camps throughout the summer, meeting its stated obligations and generating revenue without interruption.

Total savings were estimated at $4 million using ThermaPure compared to traditional remove and replace remediation, which would have closed the facilities to summer use and required extensive tear down and rebuild expenditure. The heat treatment simultaneously eradicated the termite infestation.

"Heat treatments like ThermaPure's are a win-win for the insurance company and property owner," says Michael Geyer, P.E., C.I.H., C.S.P. "Heat is even being used to achieve final clearance on tough traditional remediation projects where typical methods often fail. It can be used to salvage moisture-damaged contents instead of disposal and can help preserve historical properties in lieu of destructive removal."

PDG Environmental, a national environmental remediation contractor, used the ThermaPure process in New Orleans after recent hurricane activity. "We used it to polish off any mold or bacteria left after traditional remediation on a commercial site that was flooded with sewage-contaminated water," said John Regan, Chairman and CEO of PDG Environmental. "It dried out the building extremely quickly and helped us meet clearance levels."

Geyer adds, "Had the heat treatment been widely used in New Orleans and other hurricane ravaged areas, buildings with minor to moderate water damage could have been rapidly rehabilitated for far less than typical remove and replace remediation."

Since ThermaPure can raise temperatures in targeted areas or entire structures to levels lethal to biological pests, it has been successfully used against mold and fungi, bacteria and viruses, insect infestations, and to improve indoor air quality by accelerating the off-gassing of odors and toxins.

Alan Forbess is President of Criterion Environmental, a full-service environmental consulting firm based in Ventura, California. He is a Registered Environmental Assessor in the State of California and a Certified Microbial Consultant with the American Indoor Air Quality Council. He has provided expert witness testimony in several legal cases and managed over 1,000 mold assessments for commercial, residential and educational properties. For more info, visit www.thermapure.com; call 805-641-9333; fax 805-648-6999; email info@thermapure.com; or write to E-Therm, Inc. at 180 Canada Larga Road, Ventura, CA 93001.

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A bnp PUBLICATION

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by Jim Holland

Turning Up the Heat

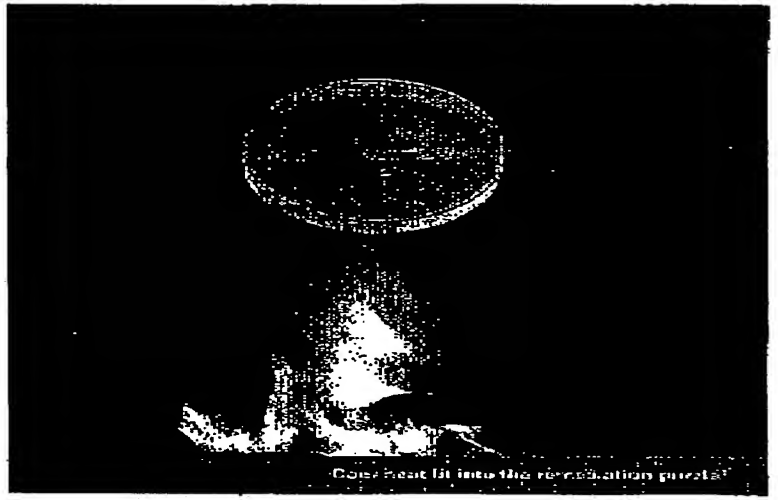
Q: I've been reading about using heat for remediation in a crawlspace. What kind of results can we get from this process?

A. First of all, I personally believe that heat is an effective method of solving bacterial problems in buildings. However, it is important to clarify what we mean by remediation. Both mold and sewage damage cleanup fall into this category. Let's begin with hot air drying in general and then discuss sewage (bacteria) and mold.

Currently there are several franchise and licensing companies that offer hot air for drying and pest control. There are also indirect fired heat exchangers that can be used for this purpose. Outdoor air is processed through the equipment where it is heated. The hot air is forced into the indoor environment or crawlspace to promote evaporation and then exhausted back to the outdoor environment. It is a process of ventilation, not dehumidification.

The indirect fired heat exchanger systems provide clean heat for drying and do not introduce combustion gases and water into the environment. The internal temperature of the heated indoor environment can range from 120 degrees to 160 degrees. Of course, in any instance where heat is used within a structure, fire safety and prevention must be considered. Also, temperatures above 160 degrees (and sometimes lower) may cause damage to certain building components or contents, so monitoring and an understanding of how building components react to heat is essential. The units are generally placed outside the building, with duct work attached to the clean air exhaust. Locating equipment outside the building may result in safety and security issues. There are other units on the market that are custom built for this purpose that have design variations.

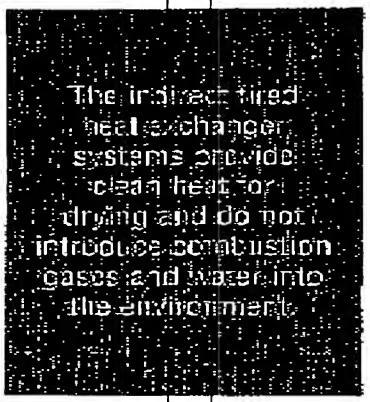
An advantage of using heat for sewage remediation in crawlspaces is the ability for heat to assist in drying the crawlspace. The elevated temperature of the air makes it "thirsty," so it has the ability to hold more moisture. This is only an advantage if the moisture-laden air is exhausted to the out-



Close heat fit into the remediation puzzle.

side. If the air isn't exhausted, but is allowed to re-circulate, the moisture may condense on cooler surfaces causing additional damage.

For years we have utilized a chart in our training classes that is derived from a study that was performed by the World Bank in 1980. It shows, among other things, that sewage-related organisms will naturally bio-deteriorate in six to 12 months if left in the soil. It also indicated that by heating the environment that sewage-related organisms (bacteria included) would die in a matter of hours.



Other options for remediating sewage in soil include soil removal and replacement, or covering the soil with a mat or polyethylene (in some cases in conjunction with depressurization using a gas mat system connected to an exhaust fan). Other options, such as using biocides or lye/lime, create other problems and have not been found to be practical or effective solutions. But soil removal is labor intensive, and bio-remediation takes considerable time to be effective. Heat, on the other hand, can speed up the process and reduce costs.

There are several issues to consider when using heat in a crawlspace. You need to ensure that the pressure differential between the crawlspace and the living area remains negative relative to the crawlspace. Studies have shown that air infiltration from crawlspaces into a structure is common. If you force air into the crawlspace, it will add pressure and increase the infiltration. That is why maintaining negative pressure in the crawlspace while drying or remediating using heat is important.

Another consideration is the depth to which the sewage has penetrated the soil. The deeper the penetration, the longer the heat is needed to raise the temperature of the soil. What is likely to occur, in most situations, is the pathogens in the top layer of soil are killed, but may remain active in cooler depths of the soil. It is also

importance to remember that even though elevated temperatures kill pathogens, the organic material remains and may still result in major odor problems over time.

The use of heat has been proposed to assist in the process of mold remediation. At this time, the research does not fully support the use of heat as a complete remediation option. Our company has conducted some preliminary research into the possible effectiveness of heat on actual mold growth. This was a preliminary study, designed to explore the ability of heat to kill mold spores and hyphae after water damage in buildings.

The study was conducted by collecting dry and wet culture swabs from previously identified mold growth of *Penicillium* and *Aspergillus*. The swabs were placed in clean sealed containers that would contain the organisms, but allow the heat to penetrate. Identical controls samples were also prepared. The controls were maintained at room temperature. The dry and wet mold samples were placed in a heated shed type building that was kept at a temperature around 160 degrees. Half of the treated samples were exposed for a period of approximately one hour. The other samples were exposed to the heat for eight hours. The results demonstrated no apparent reduction in the levels of fungal growth between the controls and the "dry" spores that were cultured after exposure to heat for either of the two treatment periods.

The "wet" spore control culture demonstrated growth consistent with that found in the "dry" spore cultures. The "wet" spore heated culture demonstrated no growth for either of the exposure times. The significance of this result is not clear since the cultures were not processed promptly after collection due to communication and shipping problems. The control samples were handled identically with the treated samples with the exception that the controls were never exposed to temperatures over room temperature. An explanation for the absence of growth from the short term and long term exposure to heat for wet samples would also require additional investigation. These same kinds of results were also observed when similar mold cultures were exposed to heat in an oven operating at approximately 170 degrees for 24 hours — there was not a significant reduction in the viability of the dry spores.

> Studies have shown that air infiltration from crawlspaces into a structure is common.

Even if heat were able to kill mold spores, it probably would still not be an acceptable technique for treating mold in crawlspace since the "dead organisms" are still problematic. The EPA in its publication "Mold Remediation in Schools and Commercial Buildings" states: "The purpose of mold remediation is to remove the mold to prevent human exposure and damage to building materials and furnishings. It is necessary to clean up mold contamination, not just to kill the mold. Dead mold is still allergenic and some dead molds are potentially toxic."

According to a recent position paper published by the "Journal of Allergy and Clinical Immunology" (Volume 117, number 2, pgs 326-333): "Allergic responses to inhaled mold antigens are a recognized factor in lower airway disease (i.e., asthma)." The position paper also states hypersensitivity pneumonitis "is an uncommon but important disease that can occur as a result of mold exposure." Both of these conditions can result from dead spores.

At present, the mechanism that causes toxicosis and the concern over inhaled mycotoxins produced by molds remains unclear. However, it has been shown that certain mycotoxins, such as satratoxin produced by *Stachybotrys*, can penetrate the skin and cause an adverse reaction. Ingestion has resulted in serious toxicity in the food industry. For this reason the food industry has significantly researched techniques to destroy mycotoxins or rendering them harmless. In "Food Safety: Foodborne Illness" it is stated that "these substances [mycotoxins] are not protein and are not destroyed by heat. The best methods of control for mycotoxins are to prevent contamination and to prevent the growth of mold." The International Crops Research Institute has stated that mycotoxins known as "Aflatoxins in dry state are very stable to heat up to the melting point." The melting point for Aflatoxins range from 237 degrees to 299 degrees. Finally, according to the Queensland Government Department of Primaries Industries and Fisheries, "Heating is not a satisfactory method for detoxifying fungal mycotoxins..."

Heat appears to be a useful tool for some applications and not others. As with any tool, it is important to learn what it can and cannot accomplish. This is obviously important to protect your company against liability and to be sure the services you offer are effective. *If you liked this article, circle 174 on the Reader Inquiry Card.* **ICS**

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Position paper

The medical effects of mold exposure

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Exposure to molds can cause human disease through several well-defined mechanisms. In addition, many new mold-related illnesses have been hypothesized in recent years that remain largely or completely unproved. Concerns about mold exposure and its effects are so common that all health care providers, particularly allergists and immunologists, are frequently faced with issues regarding these real and asserted mold-related illnesses. The purpose of this position paper is to provide a state-of-the-art review of the role that molds are known to play in human disease, including asthma, allergic rhinitis, allergic bronchopulmonary aspergillosis, sinusitis, and hypersensitivity pneumonitis. In addition, other purported mold-related illnesses and the data that currently exist to support them are carefully reviewed, as are the currently available approaches for the evaluation of both patients and the environment.
(J Allergy Clin Immunol 2006;117:326-33.)

Key words: Mold, fungi, hypersensitivity, allergy, asthma

Abbreviations used
ABPA: Allergic bronchopulmonary aspergillosis
CRS: Chronic rhinosinusitis
HP: Hypersensitivity pneumonitis
MVOC: Volatile organic compound made by mold
VOC: Volatile organic compound

Exposure to certain fungi (molds) can cause human illness. Molds cause adverse human health effects through 3 specific mechanisms: generation of a harmful immune response (eg, allergy or hypersensitivity pneumonitis [HP]), direct infection by the organism, and toxic-irritant effects from mold byproducts. For each of these defined pathophysiologic mechanisms, there are scientifically documented mold-related human diseases that present with objective clinical evidence of disease. Recently, in contrast to these well-accepted mold-related diseases, a number of new mold-related illnesses have been hypothesized. This has become a particular issue in litigation that has arisen out of unproved assertions that exposure to indoor molds causes a variety of ill-defined illnesses. Many of these illnesses are characterized by the absence of objective evidence of disease and the lack of a defined pathology and are typically without specificity for the involved fungus–fungal product purported to cause the illness.

In this position paper we will review the state of the science of mold-related diseases and provide interpretation as to what is and what is not supported by scientific evidence. This is important for members of the allergy–clinical immunology community, who are frequently asked by patients, parents, and other interested parties to render opinions about the relationship of mold exposure to a variety of patient complaints. Given the nature of this document, key rather than exhaustive citations are provided. The latter can be found in documents such as the Institute of Medicine reports "Damp indoor spaces and health"[1] and "Clearing the air: asthma and indoor air exposure."[2]

THE RELATIONSHIP OF MOLDS TO ALLERGY AND ASTHMA

It is estimated that approximately 10% of the population have IgE antibodies to common inhalant molds.[3] About half of these individuals (5% of the population) are predicted to have, at some time, allergic symptoms as a consequence of exposure to fungal allergens.[4] Although indoor fungal allergen exposure occurs, outdoor exposure is generally more relevant in terms of sensitization and disease expression. The role of indoor fungi in the pathogenesis of allergic disease has been extensively reviewed in recent reports from the Institute of Medicine of the National Academy of Science.[1]

Sensitization to fungi, particularly *Alternaria alternata*, has been linked to the presence, persistence, and severity of asthma.[5] Exposure to atmospheric fungal spores

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(principally in the outdoor environment) has been related to asthma symptoms and medication use in children with asthma.[6]

The association of asthma symptoms and exposure to indoor fungi is less clearly established. Literature reviews suggest that children living in damp houses, homes with visible mold growth, or both were more likely to experience lower respiratory tract symptoms of cough and wheeze than children who do not.[7,8] Recent prospective epidemiologic studies have shown that infants at risk for asthma, defined by a maternal history of asthma, who are exposed to high concentrations of indoor fungi (in addition to cockroach allergen and nitrogen dioxide sources) in the first year of life are at risk for persistent wheezing and cough.[9,10] These and similar epidemiologic reports fall short of prospective studies that control for confounding factors, such as humidity and other airborne allergens and irritants.

Molds are often presumed to be an important cause of the other atopic manifestations, including allergic rhinitis and, to a far lesser degree, atopic dermatitis. Abundant published data have established that sensitization (by skin testing, circulating IgE antibodies, or both) to one or more airborne molds occurs in these diseases, although sensitization is less frequent to molds than to pollens, animal danders, and house dust mite.

Current studies do not conclusively demonstrate a causal relationship of airborne mold exposure and clinical manifestations of allergic rhinitis. The data on outdoor molds (eg, *Alternaria* species and basidiomycetes) purportedly causing allergic rhinitis are indirect and conflicting.[11-13] Studies attempting to correlate indoor molds with symptomatic allergic rhinitis are even more problematic because of such methodological uncertainties as lack of quantitative mold sampling[14-16] and inclusion of upper respiratory tract infections.[17]

Published reports document mold sensitivity in some children and adults with atopic dermatitis.[18-20] However, there are no publications that establish a causal role for airborne molds in this disease rather than the IgE antibodies simply reflecting an expected concomitant of their atopic state. There are no credible reports in the medical literature documenting indoor exposure to molds as a cause of the nonatopic IgE-mediated diseases (eg, urticaria-angioedema and anaphylaxis).

Conclusions:

- Atopic patients (those with allergic asthma, allergic rhinitis, and atopic dermatitis) commonly have IgE antibodies to molds as part of polysensitization.
- Allergic responses to inhaled mold antigens are a recognized factor in lower airway disease (ie, asthma).
- Currently available studies do not conclusively prove that exposure to outdoor airborne molds plays a role in allergic rhinitis, and studies on the contribution of indoor molds to upper airway allergy are even less compelling.
- Exposure to airborne molds is not recognized as a contributing factor in atopic dermatitis.
- Exposure to airborne molds is not recognized as a cause of urticaria, angioedema, or anaphylaxis.
- Patients with suspected mold allergy should be evaluated by means of an accepted method of skin or blood testing for IgE antibodies to appropriate mold antigens as part of the clinical evaluation of potential allergies.

ALLERGIC BRONCHOPULMONARY ASPERGILLOSIS AND SINUSITIS

Allergic bronchopulmonary aspergillosis (ABPA) is a well-recognized clinical entity affecting individuals with asthma or cystic fibrosis.[21] A variety of fungi in addition to *Aspergillus fumigatus* can produce a similar clinical picture. The critical element in ABPA is an underlying anatomic change in the lung and not a specific mold exposure because at-risk individuals will have ongoing exposures caused by the ubiquitous nature of the fungi involved. Exposure to *A fumigatus* can occur both from indoor and outdoor sources.

Allergic fungal sinusitis is similar to ABPA in that it is a localized hypersensitivity condition resulting from fungal growth in an area of abnormal tissue drainage.[22] Although originally attributed primarily to *A fumigatus*, other fungi, particularly mitosporic (formerly known as Deuteromycetes or imperfect fungi) fungi are more commonly implicated (eg, *Curvularia* and *Bipolaris* species). Almost uniformly there is allergic sensitization to multiple allergens, including the fungus implicated in the affected sinus. Criteria for this condition have been well delineated, and it is generally readily distinguishable from typical chronic sinusitis. Specific criteria for diagnosis include eosinophilic mucus demonstrating noninvasive fungi, type 1 hypersensitivity (history, positive skin test result, or positive *in vitro* test result to allergens), nasal polyposis, and characteristic radiographic findings.

It has recently been proposed that most cases of chronic rhinosinusitis (CRS) are attributable to sensitivity to fungi. In particular, *Alternaria* species were suspected because most patients had these organisms recovered by means of culture from sinus surgery specimens. However, these organisms are frequently recovered from the nasal cavities of healthy individuals. Although some evidence for an immune response to these fungi in patients with CRS has been presented, clear-cut evidence for this as the cause of CRS is still lacking, and treatment with intranasal antifungal agents (eg, amphotericin) has not been conclusively demonstrated to be an effective treatment.[23]

Conclusions:

- ABPA and allergic fungal sinusitis are manifestations of significant hypersensitivity to fungi, particularly *Aspergillus* species.
- Data supporting the role of fungi in CRS are lacking at this time.

HYPERSENSITIVITY PNEUMONITIS

HP, also referred to as extrinsic allergic alveolitis, is a disease that exists in acute, subacute, and chronic forms but with considerable overlap. It is an allergic disease in which the allergen is inhaled in the form of an organic dust of bacterial, fungal, vegetable, or avian origin. Both sensitization and the elicitation of the disease state generally require high-dose exposure, prolonged exposure, or both to the causative allergen. Many cases are, in fact, occupational because of this. There are reports of a similar, if not identical, disease from workers exposed to inhaled chemicals, especially isocyanates. A few instances of the disease have been attributed to systemically administered drugs.

HP is rare, and most cases have been reported in certain occupations, such as farming, and in hobbyists, such as persons who raise pigeons. It is not a reportable disease, and therefore prevalence and incidence are difficult to estimate. The immunopathogenesis of the disease is believed to be cell-mediated (delayed) hypersensitivity. Allergen-specific precipitins are often present in serum and are important is establishing exposure. Precipitins might also play a role in the mechanism of the acute phase of the disease. HP results in acute episodes of noninfectious, immunologically mediated interstitial pneumonitis (ie, alveolitis), which might eventually produce restrictive irreversible lung disease.

The diagnosis requires a clinical and environmental history, relevant physical examination findings, chest radiography or computed tomographic scanning, high-resolution computed tomographic scanning, pulmonary function testing, bronchoalveolar lavage, and transbronchial or open lung biopsy. Specific diagnosis of the responsible allergen is performed by testing for IgG antibody to the allergen extract, typically by testing for the presence of precipitins in the Ouchterlony double-diffusion assay. In some instances provocation inhalation challenge to the suspected allergen extract might be necessary to replicate pertinent clinical and laboratory responses. Finally, a favorable response to the elimination of the inhaled antigen, administration of prednisone, or both is confirmatory. Because a differential diagnosis covers a number of respiratory diseases, an accurate diagnosis of HP demands that the clinical diagnosis be ensured.

Exposure to domestic specific indoor fungal spores is an extremely unlikely cause of HP, except in highly unusual circumstances, such as workplace exposure.

Conclusions:

- HP is an uncommon but important disease that can occur as a result of mold exposure, particularly in occupational settings with high levels of exposure.

INFECTION

Superficial mold infections (eg, tinea cruris, onychomycosis, and thrush) are common in healthy individuals and result primarily from local changes in the cutaneous or mucosal barrier, resident microflora, or both.[24,25] These infections are not the result of environmental exposure, except occasionally as related to certain animal vectors. Indeed, molds of the *Malassezia* genus are resident on the vast majority of human subjects and only become evident as "tinea versicolor" during periods of more exuberant growth. A limited number of molds (eg, coccidiomycosis, histoplasmosis, and blastomycosis) are aggressive pathogens in otherwise healthy persons. Acquisition of these infections is generally related to specific outdoor activities-exposures. Individuals with recognized primary and secondary immunodeficiency disorders are at increased risk for infection with a wide range of opportunistic fungi, with the risk varying with the degree and nature of the specific immunodeficiency. Opportunistic fungal infections are typically associated with cellular rather than (isolated) humoral immunodeficiencies. Generally, host factors, rather than environmental exposure, are the critical factor in the development of opportunistic mold infection in immunocompromised individuals because exposure to potential fungal opportunistic pathogens (eg, *Aspergillus* species) is ubiquitous in normal outdoor and indoor environments. Accepted histologic and microbiologic methods should be used to make the diagnosis of fungal infection.

Conclusions:

- Common superficial fungal infections are determined by local changes in the skin barrier, resident microflora, or both.
- A very limited number of aggressive fungal pathogens can be acquired through specific outdoor exposures.
- Host factors, rather then environmental exposure, are the main determinant of opportunistic fungal infection.

TOXIC EFFECTS OF MOLD EXPOSURE

Ingestion

Ingestion of mycotoxins in large doses (generally on the order of a milligram or more per kilogram of body weight) from spoiled or contaminated foods can cause severe human illness.[26] Toxicity from ingested mycotoxins is primarily a concern in animal husbandry, although human outbreaks do occur occasionally when starvation forces subjects to eat severely contaminated food. Specific adverse effects from a given toxin generally occur in a narrower and better-defined dose range than for immunologic or allergic effects that might vary across much broader dose ranges. Some mycotoxins, such as ocratoxins and aflatoxins, are commonly found in food stuffs, including grain products and wines, and peanut products, respectively, such that there are governmental regulations as to the amounts of allowable aflatoxin in foods.[27,28] Acute high-intensity occupational exposures to mixed bioaerosols have given rise to a clinical picture called "toxic dust syndrome." The nature of the responsible agent or

agents in that condition remains undefined, and the observed adverse effects reported have been transient. Such exposures are highly unlikely in nonoccupational settings.

Toxicity caused by inhalation

The term *mold toxicity* as used here refers to the direct injurious effects of mold-produced molecules, so-called mycotoxins, on cellular function. Toxicity should not be used to refer to changes related to innate immune responses (eg, nonspecific inflammation caused by mold particulates) or to adaptive immune responses (eg, induction of IgE or IgG antibodies). Mycotoxins are low-molecular-weight chemicals produced by molds that are secondary metabolites unnecessary for the primary growth and reproduction of the organisms. In-depth review of the toxicology of mycotoxins and their potential for adverse health effects can be found elsewhere.[1,2] It is important to emphasize key principles of toxicology relevant to patient concerns about possible toxic effects from mold exposure.

Only certain mold species produce specific mycotoxins under specific circumstances. Importantly, the mere presence of such a mold should not be taken as evidence that the mold was producing any mycotoxin. For a toxic effect to occur in a subject, (1) the toxin must be present, (2) there must be a route of exposure, and (3) the subject must receive a sufficient dose to have a toxic effect. In the nonoccupational setting the potential route of exposure is through inhalation. Mycotoxins are not volatile and, if found in the respirable air, are associated with mold spores or particulates. They are not cumulative toxins, having half-lives ranging from hours to days depending on the specific mycotoxin. Calculations for both acute and subacute exposures on the basis of the maximum amount of mycotoxins found per mold spore for various mycotoxins and the levels at which adverse health effects are observed make it highly improbable that home or office mycotoxin exposures would lead to a toxic adverse health effects.[1,29]

Thus we agree with the American College of Occupational and Environmental Medicine evidence-based statement and the Institute of Medicine draft, which conclude that the evidence does not support the contention that mycotoxin-mediated disease (mycotoxicosis) occurs through inhalation in nonoccupational settings. Furthermore, the contention that the presence of mycotoxins would give rise to a whole panoply of nonspecific complaints is not consistent with what is known to occur; when a toxic dose is achieved (eg, through ingestion of spoiled foods), there is a specific pattern of illness seen for specific mycotoxins.

Conclusions:

- The occurrence of mold-related toxicity (mycotoxicosis) from exposure to inhaled mycotoxins in nonoccupational settings is not supported by the current data, and its occurrence is improbable.

IRRITANT EFFECTS OF MOLD EXPOSURE

The Occupational Health and Safety Administration defines an irritant as a material causing "a reversible inflammatory effect on living tissue by chemical action at the site of contact." Irritant effects are dose related, and the effects are transient, disappearing when the exposure has decreased or ceased.

Molds produce a number of potentially irritating substances that can be divided into volatile organic compounds (VOCs) and particulates (eg, spores, hyphae fragments, and their components). The threshold level of irritant response depends on the intrinsic properties of the specific material involved, the level plus length of exposure, and the innate sensitivity of the exposed tissues (eg, the skin versus nasal mucosa).

VOCs made by molds (MVOCs) are responsible for their musty odor. MVOCs include a wide range of alcohols, ketones, aldehydes, esters, carboxylic acids, lactones, terpenes, sulfur and nitrogen compounds, and aliphatic and aromatic hydrocarbons.[30] Although levels causing irritant effects have been established for many VOCs, MVOC levels measured in damp buildings are usually at a level so low (on the order of nanograms to micrograms per cubic meter) that exposure would not be expected to cause complaints of irritation in human subjects.[31] Because there are other sources of VOCs indoors, measurement of indoor airborne concentrations of MVOCs is rarely done.

Mold particles (spores, hyphal fragments, and their structural components) are not volatile. These structural mold compounds (particulates) have been suggested to cause inflammation through deposition on mucus membranes of their attached glucans and mannans. However, whether such effects occur clinically remains unproved. In fact, subjects exposed to airborne concentrations of between 215,000 and 1,460,000 mold spores/m^3 at work showed no differences in respiratory symptoms at work versus while on vacation nor was there evidence of increased inflammatory markers in their nasal lavage fluids related to their mold exposure at work.[32] Thus mold particulates generally found indoors, even in damp buildings, are not likely to be irritating.

It should be emphasized that irritant effects involve the mucus membranes of the eyes and upper and lower respiratory tracts and are transient, so that symptoms or signs persisting weeks after exposure and those accompanied by neurologic, cognitive, or systemic complaints (eg, chronic fatigue) should not be ascribed to irritant exposure.

Conclusions:

- The occurrence of mold-related irritant reactions from exposure to fungal irritants in nonoccupational settings are theoretically possible, although unlikely to occur in the general population given exposure and dose considerations.
- Such irritant effects would produce transient symptoms-signs related to the mucus membranes of the eyes and upper and lower respiratory tracts but would

not be expected to manifest in other organs or in a systemic fashion.
- Further information about thresholds for irritant reactions in at-risk populations is needed to better define the role of molds, mold products, and other potential irritants in such individuals.

IMMUNE DYSFUNCTION

The question has been raised as to whether mold or mycotoxin exposure can induce disorders of immune regulation. At this time, there is no credible evidence to suggest that environmental exposure to molds or their products leads to a state of clinically significant altered immunity expressed as either immunodeficiency or autoimmunity. The published literature in this regard is of particularly poor quality and should not be relied on as scientifically valid.[33,34] Individuals who have had intense occupational mold exposures do not manifest opportunistic infections or other findings of immunodeficiency, and thus even the most intense form of airborne mold exposure is not a recognized cause of secondary immunodeficiency in human subjects. Some mycotoxins are immunosuppressive and used for this purpose clinically (eg, cyclosporine). However, the doses involved are not relevant to what might have been found in the environment. Doses that might be seen in environmental exposures are discussed in other sections of this article (toxicity and environmental sections). Testing of a wide range of nonspecific immunologic parameters, such as immunophenotyping of lymphocytes beyond those parameters having known clinical utility (eg, total B and CD3, CD4, and CD8 cells) or measurement of serum cytokines is not appropriate for assessing subjects for immunodeficiency in general and for mold-induced immune dysregulation specifically.[35]

There is also no reliable evidence for mold exposure in any setting being a linked to the induction of autoimmune diseases in human subjects. Although certain viral and bacterial infections appear to have a relationship to the induction-precipitation of autoimmune diseases, such an association has not been established for any known mold exposure. The measurement of clinically useful tests of autoimmunity (eg, antinuclear antibody), much less testing of a broad array of nonvalidated autoantibodies (eg, putative antibodies to central or peripheral myelin), is not indicated, and such testing should not be used to indicate mold exposure or mold-related disease.

This practice of testing many nonvalidated immune-based tests, as has been done previously to suggest an immunologic basis for idiopathic environmental intolerance (multiple chemical sensitivity), is expensive and does not provide useful information that will be of benefit in diagnosis, management, or both of disease and is to be discouraged.

Conclusions:

- Exposure to molds and their products does not induce a state of immune dysregulation (eg, immunodeficiency or autoimmunity).
- The practice of performing large numbers of nonspecific immune-based tests as an indication of mold exposure or mold-related illness is not evidence based and is to be discouraged.

LABORATORY ASSESSMENT

Patient assessment

Measurement of IgE antibodies to mold proteins. Assessment for IgE antibodies to mold antigens has clearly been validated as a measure of potential allergic reactivity to mold. This assessment can be done through either *in vivo* or *in vitro* testing. The relative strengths of these different forms of testing have been reviewed recently.[36,37] In general, there is a weaker correlation between *in vivo* and *in vitro* testing for IgE antibodies to mold antigens than for other antigens, partly as a result of the heterogeneity of extractable mold proteins. A positive IgE antibody level to mold proteins without appropriate clinical evaluation should not necessarily be taken as an indicator of clinical disease. In addition, the presence of IgE antibodies to a mold cannot be used to determine the dose or timing of prior exposures. Although IgE antibodies to *Stachybotrys* species can be detected through *in vitro* or *in vivo* testing, such testing should be discouraged. *Stachybotrys* species is unlikely to be a relevant clinical allergen in human subjects because it is poorly aerosolized and far less common than other well-established mold allergens.

Measurement of IgG antibodies to mold proteins. Assessment of IgG antibodies to mold proteins can be performed through immunoprecipitation–double diffusion or solid-phase immunoassays.[37] Such testing has demonstrated value in assessment of individuals with suspected HP or allergic bronchopulmonary mycosis. Immunoprecipitation assays have been classically used for the assessment of HP, and although they measure all classes of antibodies present, they are primarily detecting IgG antibodies. Such testing (immunoprecipitation or solid-phase IgG testing) is appropriate to perform only in the setting of a clinical picture, including history, physical examination, imaging studies, and other laboratory assessments, suggesting HP or allergic bronchopulmonary mycosis as part of the differential diagnosis. Use of these tests as screening procedures for these diseases in the absence of an appropriate clinical picture is discouraged.

Immunoprecipitation testing remains the standard approach because the presence of precipitating antibodies is strong supportive evidence in the appropriate clinical setting. However, as many as half of highly exposed individuals might have precipitating antibodies in the absence of any clinical disease. Solid-phase immunoassays have not been widely used for the specific diagnosis of these diseases. Although newer assays are quantitative, the actual level of IgG antibody that would be associated with either HP or ABPA has not been defined. Therefore a level of mold antigen–specific IgG antibody above a statistically defined reference range cannot be taken as evidence for HP or ABMA with the same strength as immunoprecipitation testing. Limited studies suggest that

the level of a specific IgG antibody that would be associated with HP could be 5-fold or greater than the upper limit of the nondiseased group reference range. Use of older-generation, semiquantitative, solid-phase immunoassays is not recommended.

Testing for IgG antibodies to mold proteins cannot be used as a surrogate to assess either the level or timing of specific mold exposures.[38] This is not surprising given the widespread occurrence of molds in the environment.

Measurement of antibodies of isotypes other than IgG (eg, IgA and IgM) to mold is not useful to assess mold exposure. However, the differential response of IgM and IgG antibodies is useful in diagnosis with specific organisms (eg, coccidioidomycosis). IgM levels have not been shown to relate to specific airborne exposures to molds in the absence of infection because mold exposure is common and generally ongoing. Measurement of IgA antibodies to airborne molds has not been shown to be related to a specific timing of exposure, and the claim that increased IgA antibodies to mold represents a more recent exposure than IgG antibodies is not supported by scientific evidence. Measurement of salivary IgA to mold as a marker of mold exposure has not been shown to have scientific validity.

Measurement of antibodies to mycotoxins. Mycotoxins are not proteins but low-molecular-weight chemicals. There is no scientific basis to support measurement of alleged naturally occurring antibodies to various mycotoxins as a marker of exposure to mycotoxins. Evidence of natural exposures from ingestion in human subjects and animals and use of these compounds in clinical medicine does not support the concept of naturally occurring antibodies. Such testing has not been validated and cannot be relied on as an indication of exposure to any mycotoxin.[39]

Conclusions:

- Measurement of antibodies to specific molds has scientific merit in the assessment of IgE-mediated allergic disease, HP, and allergic bronchopulmonary mycosis.
- Measurement of antibodies to molds cannot be used as an immunologic marker to define dose, timing, and/or location of exposure to mold antigen inhalation in a noninfectious setting.
- Testing for antibodies to mycotoxins is not scientifically validated and should not be relied on.

Measurement of molds and mold product exposure in the patient's environment

An in-depth analysis of methods to measure fungal organisms, mold products, and mycotoxins in the environment is outside the bounds of this article. Such information is reviewed in depth elsewhere.[40,41]

Measurement of fungi in the subject's environment. Measurement of airborne fungal spores in the subject's environment by using culture methods, nonculture methods, or both is commonly used. Air testing provides the most relevant measure of exposure and is usually reported as colony-forming units or spores per cubic meter of air. However, this testing suffers from the drawback that it is a snapshot that does not integrate exposure over time and provides data only about the location of sampling. Indoor testing must be compared with outdoor testing and preferably with more than one outdoor sample. Currently there are no standards as to what constitutes acceptable levels of outdoor or indoor airborne fungal spores.

Given these caveats, the levels of airborne fungal spores found in an indoor setting can be considered in relative and absolute terms. Indoor fungal spores arise from outdoor sources present within soil and vegetation. Therefore an increase in indoor-outdoor concentrations of specific fungi indicates the presence of an indoor source. Depending on clinical or other indications, it might be necessary to locate the source and, if necessary, take appropriate action. Total fungi spores that are greater in concentration in indoor than outdoor air might be potential evidence of increased fungal presence indoors. However, in normal indoor environments xerophillic fungi, such as *Aspergillus* and *Penicillium* species, might be found indoors at levels above those measured outdoors on a given day. Even when the fungal levels are greater indoors than those outdoors, health risks would be limited in most cases, except to the subject specifically allergic to the mold in question. Absolute fungal spore levels indoors can be put into context when one realizes that outdoor levels can reach tens of thousands of fungal spores per cubic meter and hundreds of thousands per cubic meter or higher around rotting vegetation compost or in agricultural settings, such as in grain elevators.

Bulk, surface, and within-wall cavity measurements of fungi, although sometimes indicating the presence of fungi, do not provide a measure of exposure. Fungi found in these places require a route of exposure through air (aerosolization and entry into the patient's respirable air) that involves many factors not included in these measurements. Such testing should not be used to assess exposure.

Measurement of fungal products in the patient's environment

Another approach to measure of potential fungal exposure is to test for fungal products in the environment.

Structural fungal materials. Testing for the levels of general mold structural material (eg, β-glucans in settled dust) has been used to try to integrate levels of potential exposure to molds in general over time. Although an interesting research avenue, such testing does not provide any information as to the nature of the specific fungi involved or their source (indoor or outdoor), is not useful for predicting health effects, and has not found general acceptance, as discussed elsewhere.

Mycotoxins. Specific molds can produce, under some conditions, a variety of mycotoxins or none at all. Thus measurements of spores cannot be used as surrogates of mycotoxin exposure. Mycotoxins can be measured directly. A variety of methodologies based on mass

spectroscopy have been applied to bulk samples with heavy fungal growth to identify the presence of mycotoxins; however, potential levels of mycotoxins in nonagricultural air samples are too low to be measured practically with this technology. The occurrence of mycotoxins in bulk sampling does not provide evidence of exposure because mycotoxins themselves are nonvolatile. Thus exposure requires inhalation of mycotoxin-containing spores or fungal fragments in the respirable air. For example, satratoxin H can be found in a sample of material with heavy *Stachybotrys chartarum* growth, but *Stachybotrys* species are not easily aerosolized. Testing with crude cytotoxicity of extracted bulk materials suffers from a lack of sensitivity and specificity. Such testing cannot be relied on to predict or evaluate health effects.

VOCs. See section on irritant effects above.

Conclusions:

- Sampling of both indoor and outdoor air for mold spores provides a measure of potential exposures and can be useful in certain clinical conditions, but it has many shortcomings.
- Bulk, surface, and within-wall cavity measurement or molds or mycotoxins, although having potential relevance for other purposes, cannot be used to assess exposure.
- Testing for airborne mycotoxins in nonagricultural environments cannot be used to diagnose mold exposure.

REMEDIATION

Issues regarding remediation of mold are beyond the scope of this article. Indoor mold growth should be addressed. These matters are reviewed at length in the Institute of Medicine 2004 report "Damp indoor spaces and health." For an overview, the reader can refer to the Occupational Health and Safety Administration document "A brief guide to mold in the workplace."[42] The true challenges of mold remediation are currently being addressed in the flood-ravaged areas struck by hurricane Katrina, which will unfortunately provide a rich environment for the study of both mold-induced disease and mold remediation.[43,44]

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AIRBORNE PARTICLE SIZES AND SOURCES FOUND IN INDOOR AIR

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Abstract—As concern about indoor air quality (IAQ) has grown in recent years, understanding indoor aerosols has become increasingly important so that control techniques may be implemented to reduce damaging health effects and soiling problems This paper begins with a brief look at the mechanics of deposition in the lungs and the aerosol dynamics that influence particles at all times This discussion shows that the particle diameters must be known to predict dose or soiling and to determine efficient mitigation techniques The particle sizes produced by the various indoor sources, as well as unusual aspects of each type of source, must be known so that this process may begin

This paper summarizes the results of a literature search into the sources, sizes and concentrations of indoor particles There are several types of indoor particles plant and animal bioaerosols and mineral, combustion and home/personal care aerosols These types may be produced indoors or outdoors, entering through building openings The sources may be short term, seasonal or continuous Particle sizes produced vary from submicrometer to larger than 10 μm The particles may be toxic or allergenic This information is presented in a summary table and is discussed in the text

Key word index Particles, indoor air aerosols, particles size, indoor sources, IAQ

INTRODUCTION

Knowledge of particle sources, sizes, concentrations, phases and compositions in indoor air is important because of the potential health effects and the problems related to deposition on surfaces This literature search has been performed to gather this information for use in designing test methodologies for air cleaners and other mitigation approaches These data will also aid in the selection of appropriate air cleaners Indoor air quality (IAQ) depends on the results of these efforts

Health implications

Health effects that result from inhaling indoor aerosols are directly related to the particle diameters and the total mass inhaled The single most important feature in lung deposition is the size of the particles Particles larger than 30 μm in aerodynamic diameter (the diameter of a unit density sphere of the same mass) have low probability of entering the nasal passages Figure 1 shows the American Conference of Governmental Industrial Hygienist's standards for particle sampling to approximate the deposition in various regions of the respiratory tract (Phalen *et al*, 1986) The rapid and sharp changes of direction of air flow occurring in the passages of the nose and pharyngeal region favor deposition of larger particles Most of the particles deposited here are 5–10 μm in diameter

In the tracheal bronchiolar region, air velocity and directional changes decrease The aerodynamic diameter range favored for deposition in this range is from 1 to 5 μm The smaller particles are distributed throughout the alveolar segments of the respiratory tract As the velocity decreases to virtually zero, more time is available for sedimentation to occur, resulting in fewer and fewer particles reaching the alveoli Gravity becomes less important as the particles become smaller, thus particles, usually smaller than 1 μm, are deposited on alveolar walls mostly by diffusion

The interaction between particles and cells is largely dependent on where in the respiratory tract the particles deposit For example, particles deposited in the alveoli require more mechanisms for removal than particles that deposit in the upper respiratory tract However, the dose received by the person is dependent on the solubility of the particles and other aspects, as well as the deposition site

Bioaerosols, including bacteria and viruses, present special health hazards due to the risk of infection as discussed in the animal aerosol section

For further information, see Casarett (1975), Gardner and Finley (1983), Hinds (1982), Knight (1980), LaForce (1986), Lippmann (1972), Nelson *et al* (1988),

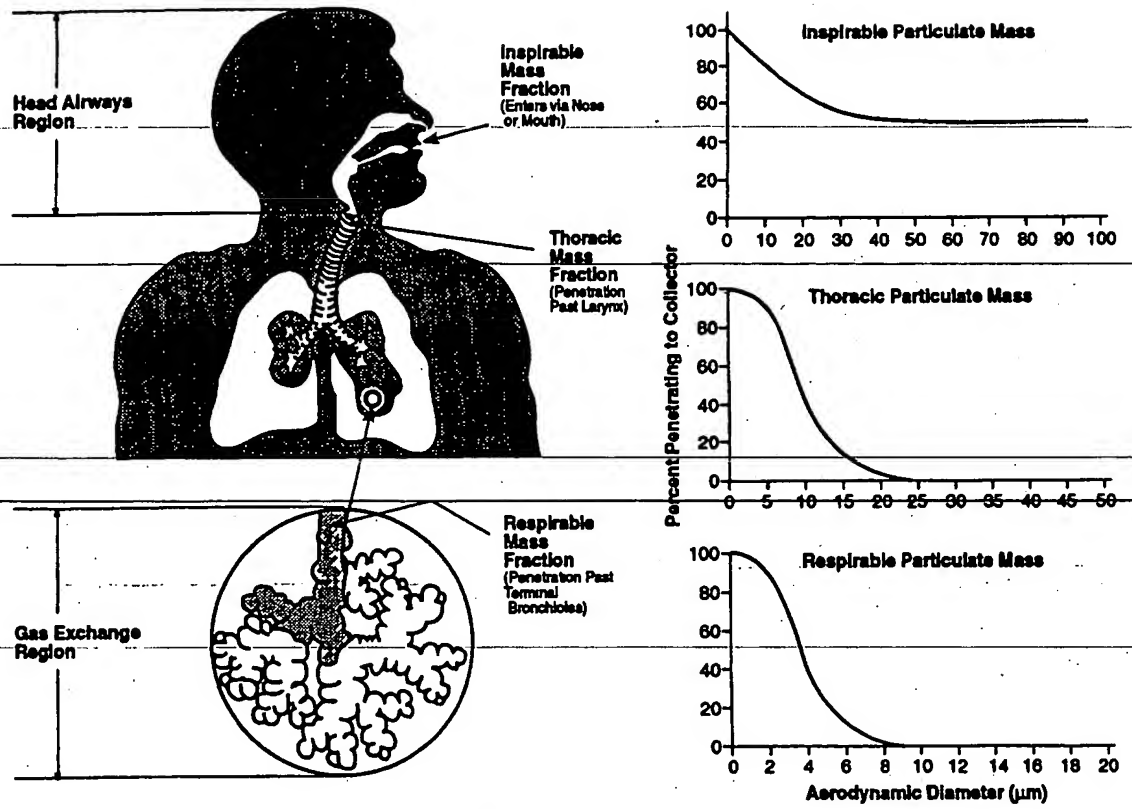


Fig 1 The three aerosol mass fractions recommended for particle size-selective sampling

Parkhurst *et al* (1988), Phalen *et al* (1986), Repace and Lowrey (1985), Revsbech *et al* (1987) and Turiel (1985)

Soiling

Problems caused by indoor aerosols, other than those due to health effects, include deposition on surfaces that results in dirty floors and windows in the home and office, failure of precision machinery, soiled and discolored art work in museums, etc Again, the particle diameters determine the path taken by the particles whether they deposit on a horizontal or vertical surface, remain in the airstream, or are removed by an air cleaning device

For more information, refer to Baer and Banks (1985), Gardner and Finley (1983), Nazaroff and Cass (1989), Okada and Matsunuma (1974), Raes *et al* (1987) and Raunemaa *et al* (1989)

Aerosol formation

General sources and mechanisms that form aerosols include condensation, combustion, nuclear degradation, resuspension and spraying Condensation of vapors in gas streams produces small liquid particles Combustion results in small liquid and solid particles, as well as larger solid particles such as soot Nuclear degradation results in ultra-small particles of radon progeny Resuspension that occurs with sweeping or in-breezes results in large solid particles reentering the air Spraying yields medium liquid or small particles

Aerosol removal

The motion of particles is determined by the kinetic properties of the gas and other external forces that act on the particles The following physical phenomena can produce forces that result in motion, transport or deposition of aerosol particles gravitational and electrical fields, drag forces, centrifugal flows, inertial forces, shear gradients, Coriolis forces, and concentration and thermal gradients Interfacial phenomena include evaporation, condensation, nucleation, adhesion and electrical charging of particles Evaporation and condensation of droplets change the size distribution of the particles Evaporation reduces or eliminates some particles, condensation leads to the growth of other particles Critical diameter is used to determine which particles will grow by condensation This diameter depends on vapor pressure Particles smaller than the critical diameter will evaporate with their mass becoming available to aid in the growth of the larger particles

Adhesion forces arise from particle and surface properties, interface geometry and condensed gas con-

stituents When small aerosol particles deposit on a solid surface, they usually adhere on contact due to these forces The adhesive force can be increased by particle electrostatic charge, but high humidity can counteract this effect Most air-cleaning devices use this property to collect particles

The electrostatic charge associated with suspended particles consists of an excess or deficiency of electrons or an excess of ions attached to the particle Most small particles have naturally acquired charges from electron transfer during contact or separation or because of free-ion diffusion Collision and adhesion of oppositely charged particles (or particles and a surface) affect sedimentation rates The maximum likely particle charge increases with particle diameter Electronic air cleaners use this property by charging particles then collecting them on opposite charged surfaces

External forces that may act on aerosol particles include gravitational, electrical, thermal and molecular forces Sedimentation, resulting from gravity, leads to particles settling out of a stream onto horizontal surfaces The settling velocity of a small spherical particle can be closely approximated by Stokes' law and is directly proportional to the particle diameter squared As the sedimentation velocity or particle size increases, inertial effects in the fluid become important and must be incorporated into the velocity calculation Once the sedimentation velocities have been determined, the rate of deposition on surface due to sedimentation alone can be calculated Sedimentation is an example of a macroscale mechanism The settling velocity, which directly relates to the number of particles of a given size that deposit, is shown by particle size in Fig 2 This figure shows that settling velocity and time to terminal velocity increase rapidly with particle size

Impaction occurs when a particle collides with an obstacle in the flow path Smaller particles follow the gas flow lines around an obstacle, whereas larger particles, owing to their greater inertia, are unable to change their direction, as shown by the time to terminal velocity in Fig 2, resulting in impact with the obstacle Thus impaction shifts the particle size distribution toward the smaller particles Filters use this method (as well as others) to capture particles

Diffusion of aerosol particles in a gas, a microscale mechanism, is the result of their bombardment by molecules of the gas (Brownian motion) Diffusion is seldom considered for particles larger than 1 μm in diameter As shown in Fig 3, the diffusion coefficient, Brownian motion and mobility decrease rapidly with increasing particle diameter Diffusion can result in the deposition of particles on surfaces in addition to that caused by sedimentation and impaction Filters use this mechanism primarily to capture small particles Figure 4 shows which forces most strongly affect the collection efficiency of a typical filter, illustrating the influence of particle size on which force is predominant and on the total collection efficiency for the filter The most penetrating size is in the region between mechanisms

In addition to these phenomena that remove particles from a stream to a surface, particles may collide with each other, due in part to diffusion, and coagulate to form larger particles This is the process primarily responsible for the removal of very small particles from the air and results in a shift in the size distribution toward the larger particle sizes

It is important to understand these forces in order to predict which particles will remain in the air long enough to be inhaled, resulting in potential health problems, and which will deposit resulting in soiling or damage to surfaces Knowledge of the sizes of the

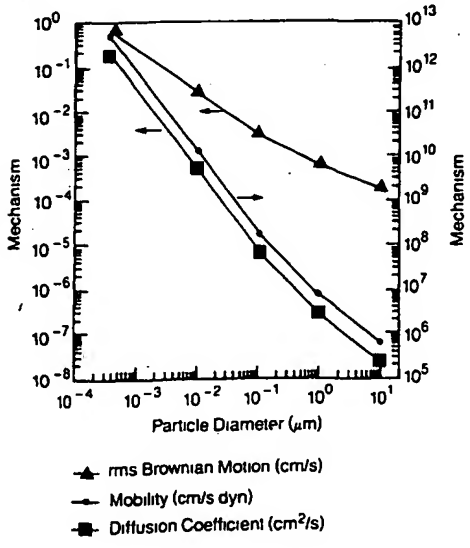


Fig 2 Microscale mechanisms

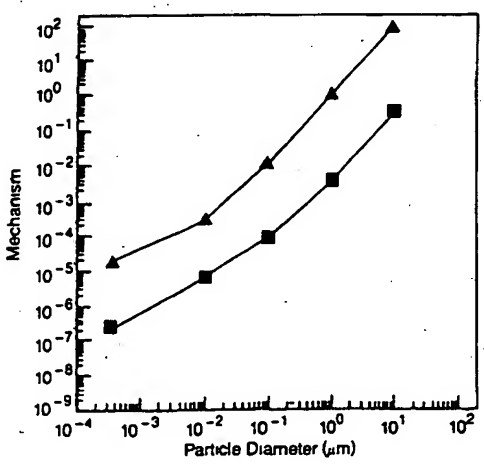
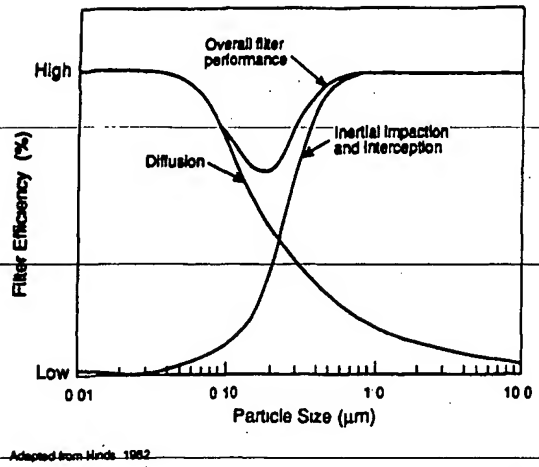


Fig 3 Macroscale mechanisms



Adapted from Hinds 1982

Fig 4 Particle removal efficiency as a function of particle size for a typical fibrous filter

particles will also enable the selection of appropriate air cleaning measures

For additional information, see Fisk *et al* (1987), Hinds (1982) and Nelson *et al* (1988)

Aerosol-producing mechanisms

Aerosols can be classified as either dispersion or condensation aerosols Dispersion aerosols are formed by mechanically breaking up a solid or liquid through such processes as grinding or atomization or by redispersing a powder Condensation aerosols are formed when vapors condense or when a gas-phase reaction produces an aerosol product In general, dispersion aerosols are larger than condensation aerosols and tend to be more polydisperse

See Fuchs and Stugin (1964) for more information

The particles in indoor air are produced or become airborne by several different mechanisms Friction between moving parts or pieces of furniture will produce solid particles, sweeping, vacuuming and dusting reentrain solid particles, and humidifiers and various sprayers produce liquid particles Smoking and cooking produce condensation aerosols, both solid and liquid In addition to particles produced by these mechanisms, other not so obvious particles, such as radon progeny, are produced through processes such as nuclear degradation

PARTICLES IN INDOOR AIR

An important approach to assessing the indoor particulate contamination problem is to identify the potential sources of indoor aerosols Then it is possible to determine the sizes, phase(s) and typical concentrations of the particles these sources produce This information is necessary to determine which types of air cleaners will be efficient in reducing the particle concentrations Also with this information, it is possible to begin calculations based on aerosol dynamics Comparison of these results to actual particle size distributions will aid in the understanding of the specifics of indoor aerosol dynamics

The following paragraphs present the results of a literature search aimed at identifying types of sources and the sizes of particles they produce Some of the particles included are larger than those normally included as aerosols However, due to the characteristics of indoor activity (e g sweeping and people moving), larger particles (e g cat hair) that will often be entrained, if only for short periods, have been included Figure 5 shows the reported size ranges for many indoor particles grouped by source type Table 1 presents a summary of the particle types and their sizes, as well as brief notes on their shapes or unusual characteristics This table includes the data used to generate Fig 5 Note that the data are reported in differing forms, as presented in the literature Spherical particles are represented as a single average diameter or by a range of diameters Other shapes are given by average dimensions or otherwise as appropriate Table 2 summarizes values reported in the literature for concentrations produced by specific sources These data are limited since most reports include total mass or total number of particles not merely those from a specified source

The sources have been classified into six types bioaerosols (plant and animal), mineral, combustion, home/personal care and radioactive aerosols

Plant aerosols

Bioaerosols contain particles of living origin, either plant or animal Plant particles include pollens, spores, molds and miscellaneous by-products Most of these particles will be of outdoor origin and will infiltrate through windows, doors, cracks and the heating, ventilating and air-conditioning (HVAC) system Of course, when plants are indoors, these particles will be produced indoors Molds will usually be present indoors, as well as outdoors Plant products such as cornstarch will be purely of indoor origin and will be readily apparent to the occupants

Pollen and spores are given off by plants at certain times of the year These particles are often too large to remain in the air for prolonged periods However, the sweeping, dusting and vacuuming that are used to remove them from floors and other surfaces reentrain a large percentage of the particles In this way even the larger particles can remain an aerosol problem for some time These types of particles present a special hazard as allergens For many people, allergic reactions greatly outweigh the potential lung deposition as a source of health concern

Molds are also a naturally occurring source of plant particles These, however, are present all year with the greatest concentrations occurring during, or immediately after, wet or humid periods (either weather or indoors due to humidifiers, etc) One potential source for high levels of indoor airborne mold is ultrasonic

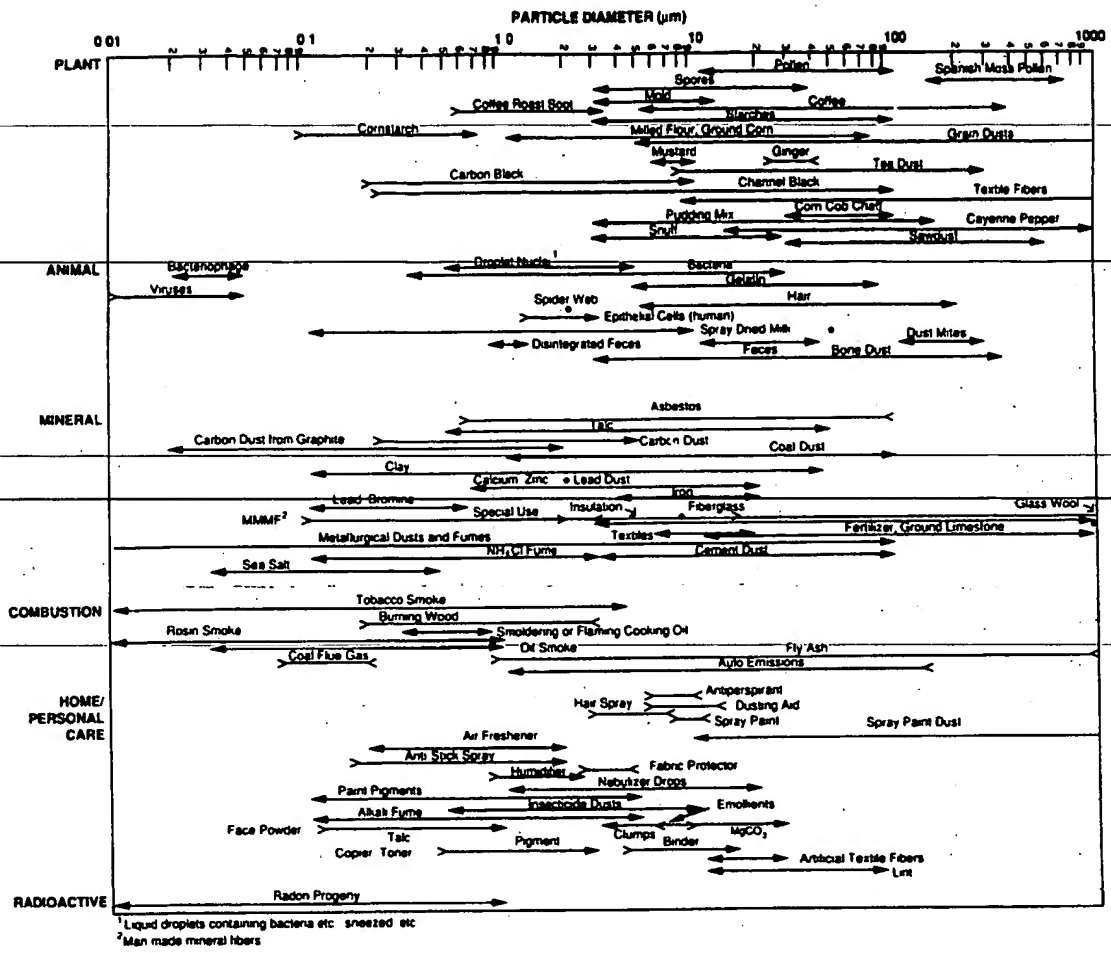


Fig 5 Sizes of indoor particles

humidifiers Molds may grow in the stagnant water left in the humidifier and then be aerosolized when the unit is reactivated Molds are also potential allergens In addition to the health problems, molds can be unsightly, will stain surfaces and may ruin furniture In humid environments mold may grow on HVAC filters, thus the air-cleaning system may exacerbate the contamination problem

See Hinds (1982) and Ishii *et al* (1979) for more information

Miscellaneous particles of plant origin come from such sources as coffee roast soot and cornstarch These particles are intentionally introduced to the indoor environment by the occupants and either intentionally or unintentionally aerosolized Cooking generates many particles Opening containers of finely ground grains results in some air contamination However, much of this will settle out rapidly and wet cloth cleanup yields less reentrainment than vacuuming or sweeping This type of source is usually confined to a small portion of the building, although the particles may be carried to the rest of the structure Nonetheless, source control or venting is much simpler than for pollens and molds

Animal aerosols

Bioaerosols are also produced by animals These particles may be very small and remain airborne for long periods or quite large and only remain in the air for short periods This type of particle includes bacteria, viruses, hair, insect parts and dandruff

Bacteria have many sources in buildings They may come from outdoors by air, in water, on shoes, with equipment, etc People transport bacteria on their clothing, as well as in their bodies Bacteria will grow indoors in many locations Bacteria become airborne through many mechanisms Since they are small, slight breezes may pick them up People aspirate them as droplet nuclei House cleaning, such as sweeping, spreads them Toilet flushing may aerosolize them Bacteria that grow in duct work, on filters or on fans are spread through the HVAC system Ultrasonic humidifiers spray them into the air

For more information, refer to Arnow *et al* (1982), Green and Lane (1964), Knight (1980) and Riley (1982)

Bacteria present a special problem since they reproduce A single bacterium or colony can grow to become a major problem Disease bacteria present a

Table 1 Sizes of particles that may occur indoors

Item	Diameter (μm) Smallest		Largest	MMD	Notes	Phase	Ref
			Plant aerosols				
Pollens					common allergens solid		
American elm	28		58		in H_2O		a
Bermuda grass	22 7		31 5				b
black walnut	28 6		40 6				b
clover				52			c
corn	75		92				b
corn				100			c
cottonwood				22			a
dandelion				25	(12–29) × (13–30)		a
general	10		100				d
horse chestnut	25	×	14				a
lamb quarters				25 8	aqueous		a
orchard grass				31 0	elliptical		c
others	26	×	14	26,34			a
paper mulberry	10 3		14 3				b
ragweed	17 8		22 2				b
				19 6	spherical		c
rye				55			c
Spanish moss	150–200	×	500–750		very thin		a
sugar maple				35			a
Spores					can be allergens	solid	
Bermuda grass smut	5 1		7 5				b
cinnamon	40		70				a
corn rust				6 8			c
fern							
fungal			200*		common allergen		e, f
general	10		30			solid	d
Johnson grass smut	5 8		9			solid	b
lycoperdon				2 09			b
lycopodium	25		35				b
lycopodium				30			c
lycopodium	31		38				a
marginal shield	40	×	25		average		a
penicillin				5			a
puff ball	4 8		8 6				c
puff ball	3		4				a
rattlesnake	18		30				a
wheat smut				4 5			a
Molds					common allergen		g
	10–12 μm wide ribbons				sporangiophores	solid	a
	3		5		spores		a
Starches							
arrowroot starch	7		75			solid	a
potato starch	15		100			solid	a
rice starch	3		30			solid	a
tapioca starch	5		25			solid	a
tea dust	<8		300			solid	a
wheat starch	3		100		can be allergen	solid	a, g
Miscellaneous general							
barley grain dust	10		380			solid	a
carbon black	<0 5		10		most 0 5–4	solid	a
cayenne pepper	15		1000			solid	a
coffee	5		375		several shapes	solid, liquid	a
coffee roast soot			4			solid	c
corncob chaff	30		100			solid	a
cornstarch			3 0		irregular	solid	c
cotton fibers	8–33	×	10–27 2 mm			solid	a
cotton linters	10–25	×	17 mm			solid	a
ginger	24		45			solid	a
ground corn	some 25 most ~900				can be allergen	solid	a, g
flax	~17 × ≤3 0 cm					solid	a
hemp	10–50 × 2 mm–3 cm					solid	a
jute	15–25 × 200 μm–8 mm					solid	a
kapok	10–35 × 2–3 cm					solid	a
milled flour	1		70			solid	d

Table 1 (contd)

Item	Diameter (μm) Smallest	Largest	MMD	Notes	Phase	Ref
mustard	6	10			solid	a
pudding mix	3	148		55% cornstarch	solid	a
sawdust	32	640			solid	a
snuff	3	25			solid	a
soybean dust	5	2000			solid	a
Animal aerosols						
Bacteria					mixed	c
E coli	2 ×	14		cylinder		c
Serratia indica	1 0 ×	2 0		cylinder		c
Serratia marcescens	1 2 ×	2 0		cylinder		c
B globigii	1 6 ×	3 0		cylinder		c
Bacteria—general	0 3	30				d
Bacteriophage					mixed	
E coli-T-3	0 02	0 05		spherical		c
Bone dust	3	385			solid	a
Droplet nuclei*	0 5	5			liquid	h
Dust mite feces	10	43	24	common allergen	solid	i
when disintegrated	0 8	1 4			solid	i
Epithelial cells (human)†			20		solid	a
Gelatin	5	90			solid	a
Hair				can be allergens	solid	
fruit bat			to 50+	1–1 5 cm long		a
Siamese cat type 1	50	70		width		a
Siamese cat type 2	25	35		width		a
dog—small	10	90				a
dog—large	10	75				a
mohair	10	90				a
wool	10	70		common allergen		a g
human	50	150				a
rabbit type 1	100			width		a
rabbit type 2	5	30	13 5			a
House dust mite	100	300			solid	i, j
Inspect parts				vary	solid	a
Spider web			~1 7	width	solid	a
Spray dried milk	0 1	10			solid	d
agglomerates			~50			a
Viruses	0 03	0 05			mixed	d
Mineral aerosols						
Asbestos		0 5		irregular	solid	c
	<1 wide fibrils make up 70+ wide bundles					a
Bromine	0 1	0 65			solid	k
Calcium	0 65	20			solid	k
Carbon dust		5 0		irregular	solid	
from graphite	0 002	2		chains of sphere	solid	c
Cement dust	3	100			solid	d
Coal dust	1	100			solid	d
Clay	0 1	40			solid	c
Fertilizer	10	1000			solid	d
Fiberglass			8	diameter	solid	a
Glass wool	3	15	10	width	solid	
Ground limestone	10	1000			solid	d
Ground talc	0 5	50			solid	d
Iron	3 6	20			solid	k
Lead	0 1	0 65			solid	k
Lead dust			2 2		solid	c
Man-made mineral fibers (MMMF)	0	2000		length and diameters	solid	l
MMMF—insulation	3	15		nominal		m
—textiles	6	20				m
—special use		<1 5				m
	5	1000	25	length		a
Metallurgical dusts and fumes	0 001	100			solid	d
NH_4Cl fume	0 1	3			solid	d

Table 1 (contd)

Item	Diameter (μm) Smallest	Largest	MMD	Notes	Phase	Ref
Saccharin		15		~sphere	solid	c
Sea salt	0.03	0 5			solid	d
Talc					solid	
micronized			25	irregular		n
coarse			16	irregular		n
Zinc	0 65	20			solid	k
		Combustion aerosols				
Auto emissions	1	120+		can be allergenic	solid, liquid	a
Burning wood	<0 3	>2 5		can be allergen	solid, liquid	g, o
Channel black	<0 5	100			solid	a
Cigarette smoke, mainstream	0 25	5		volumetric modes	solid, liquid	p
Flaming Xmas tree			0 18–0 37		solid, liquid	q
Flaming cooking oil			0 8–0 4		solid, liquid	q
Fly ash	1	200		spherical and	solid	d
Fly ash	<1	2000		irregular	solid	n
Oil smoke	0 03	1			solid, liquid	d
Pulverized coal utility boiler flue gas			0 16–0 16		solid, liquid	r
Rosin smoke	0 01	1			solid, liquid	d
Smoldering cook oil			0 8–0 35	common allergen	solid, liquid	q
Tobacco smoke	0 01	1		can be allergen	solid, liquid	d, g
Wood burning in fireplace hard, softwood, fake			0 17		solid, liquid	s
		Home/personal care aerosols				
Antiperspirant				can be allergen		g
during spray			6 8–8 11		liquid	t
persistent			5 9–7 27		liquid	t
Dusting aid						
during spray			8 4–12 4		liquid	t
persistent			6 4–7 5		liquid	t
Hair spray						
during spray			2 8–3 4		liquid	t
persistent			4 5–6 2		liquid	t
Paint				can be allergen		g
during spray			8 1–9 7		liquid	t
persistent			7 1–8 7		liquid	t
Acetate	20	30		width	solid	a
Acrylic	20	30		width	solid	a
Air freshener	0 2	2	~1 7	can be allergen	liquid	g, u
Alkali fume	0 1	5			solid	d
Anti-stick spray						
1 min after	0 55	2	~1 7		liquid	v
40 min after	0 45	1 9	~1 6		liquid	v
90 min after	<0 2	1 8	~0 9		liquid	v
Fabric protector			2 6–4		liquid	t
Face powder				can be allergenic		g
mixture of		~1		talc 75%	solid	a
	3	27		small clumps		
	5	30		$MgCO_3$		
	6	8		emollients		
Humidifier			<2 5	can carry allergens	liquid	g, v
Insecticide dusts	0 5	10			liquid	d
Lint plant, animal and man-made fibers	10	90			solid	a
Nebulizer drops	1	20			liquid	d
Nylon, bright	20	30		width	solid	a
Nylon, semidull			15	width	solid	a
Paint pigments	0 1	5			solid	d
Paint spray dust				can be allergen		g
individual spheres	8	100			solid	a
clumps	50	1000+			solid	a
Photocopier toner				can be allergenic		g
	≤15			binder		a

Table 1 (contd)

Item	Diameter (μm) Smallest	Largest	MMD	Notes	Phase	Ref
	<0 5	3		pigment	solid	a
Polyester	10	15		width	solid	a
Rayon			11	width	solid	a
Rayon, viscous	10	50		width	solid	a
Radioactive aerosols						
Radon progeny	0 005	0 4			solid	w
	0 001	1 0				x

* Droplet nuclei produced by coughing, sneezing and talking carry the infectious organisms

† Dandruff is one or more epithelial cells

(a) McCrone and Delly (1973), (b) Duke Scientific Corporation (1985), (c) Girman *et al* (1982), (d) Hinds (1982) (e) Burge and Solomon (1987), (f) Ishii *et al* (1979), (g) Faelten *et al* (1983), (h) LaForce (1986), (i) Anderson and Korsgaard (1986), (j) Academic American Encyclopedia (1988), (k) Flocchini (1977), (l) Turiel (1985), (m) Riley (1982), (n) Dennis (1976), (o) Raes *et al* (1987), (p) Chang *et al* (1985), (q) Krafthefer and Lee (1984), (r) McElroy *et al* (1982), (s) Dasch (1982), (t) Mokler *et al* (1979b) (u) Gardner and Finley (1983), (v) Highsmith *et al* (1988), (w) Walsh *et al* (1984) (x) Parkhurst *et al* (1988)

problem in addition to that of nonliving aerosol since they may cause illness Droplet nuclei are in the size range shown to have increased infectivity when aspirated Bacterial infection may spread through an entire building through the very equipment intended to purify the air However, in many cases bacteria can be controlled using standard disinfectants—a disinfected bathroom spreads fewer bacteria than one that is neglected Bacteria may attach to other particles and be transported with them

Another animal aerosol is viruses The sources and problems that are explained above for bacteria apply to viruses as well However, viruses are much smaller than bacteria, will stay airborne longer and will be less likely to be caught by filters In addition, some disinfecting methods that would be effective against many bacteria will not kill viruses

See Brundage *et al* (1988) and Knight (1980)

The next category in the summary table is that of hair Although most hairs are too big to remain aerosols for extended periods, they will be in the air at least occasionally Hairs are produced by many animals including pets and humans They become airborne as a result of falling out, trimming and brushing Again certain types of cleaning, such as sweeping, cause these particles to become reentrained after settling out of the air stream In addition to inhalation problems, hairs are important from the allergy and soiling perspectives However, longer hairs are relatively easy to collect and will become trapped in a standard vacuum cleaner or on a filter if they remain airborne long enough

Epithelial (skin) cells flake off humans and animals Dandruff is simply two or more epithelial cells clumped together These cells are shed as a normal part of growth After they are shed, they may become airborne or remain on a surface The aerosolized particles may settle out and become room dust or stay in the air as inhalable particles

Insects and arachnids (the family that includes spiders) also produce particles Insect parts and by-products can become aerosol particles These animals and their by-products may come from outdoors In the case of smaller arachnids, such as mites, the source is indoor infestation, usually in upholstered furniture, beds and dusty corners These particles enter the air through windows, breezes and household cleaning Problems presented by this type of particle include allergic reactions and soiling These particles may carry bacteria or viruses that lead to disease

One example of this, and one of the most talked about sources of particles at this time, is the house dust mite The house dust mite itself is too large to be readily airborne although its parts may be The house dust mite's feces are considered to be a major source of the allergic reaction some people have to indoor dust The fecal pellets disintegrate to form particles in the respirable range The most commonly recommended methods to reduce exposure to this allergen do not involve air cleaners Reduction of household humidity to 45% relative humidity or less is recommended to control the growth of mites, but the most stressed methods for reductions are frequent cleaning and removal of breeding grounds For persons with this allergy, removal of all rugs and carpets, covering beds with plastic sheets, frequent changing of bed linen and frequent floor cleaning, etc, are recommended However, literature discussing the effectiveness of air-cleaning devices in controlling the allergic reaction was not found

For more information see Ishii *et al* (1979)

Mineral aerosols

Mineral aerosols are produced when nonorganic matter is broken down by natural processes such as weathering or artificial processes such as grinding Many of these particles are produced outdoors and enter through windows and cracks or are brought

Table 2 Concentrations and source rates for particles

Item	Concentration range ($\mu g\,m^{-3}$)* Lower	Upper	Median	Source rate ($\mu g\,h^{-1}$)* Lower	Upper	Median	Ref
			Plant aerosols				
Mold total			742 colony forming units/m m^3				a
Cladosporium			456 colony forming units/m m^3				a
Penicillium			108 colony forming units/m m^3				a
Aspergillus			22 colony forming units/m^3				a
			Animal aerosols				
Not found							
			Mineral aerosols				
Calcium	0 110	0 400					b
Man-made mineral fibers (MMMF)	0	356					c
during installation	5×10^{-3}	0 4 fibers cm^{-3}					d
before	1×10^{-3}	0 03 fibers cm^{-3}					d
			Combustion aerosols				
Cigarette	3×10^{8}	3×10^{10} cm^{-3}		8 4	67	30 mg/cig	e,f
Wood-burning in fireplace							
hard, softwood, fake				2 1	20 g kg^{-1}		g
Kerosene heater				30	160		
			330			9×10^{11} min^{-1}	h
Wood heater	27	91		3	50 g kg^{-1}		i, j
Wood stove, airtight		<290	11–36				k†
Wood stove, not airtight		$<1 \times 10^{4}$	210–970				k
Gas oven				<0 05 μg kJ^{-1}		(8400 kJ h^{-1})	k
Gas top burners			(9200 kJ h^{-1})	0 24	0 6 μg kJ^{-1}		k
Gas space heater				0 02	0 3 μg kJ^{-1}		l

Quartz heater			31			
Heavy coil heater			16		25×10^{9} min^{-1}	h
Fine coil heater					40×10^{9} min^{-1}	h
					15×10^{10} min^{-1}	h
			Home/personal care aerosols			
Antiperspirant						
during spray			172±95 mg m^{-3}			m
persistent			246±53 mg m^{-3}			m
Air freshener			27±3 mg m^{-3}			m
Copy machine	50	500				n
Dusting aid						
during spray			100+58 mg m^{-3}			o
persistent			86+9 mg m^{-3}			o
Fabric protector			9+3 mg m^{-3}			m
Furniture wax		15±2	22 mg m^{-3}			m
Hair spray						
during spray			18±4 mg m^{-3}			o
persistent			18±2 mg m^{-3}			o
Humidifier–whole house						
impeller	56	191				p
steam			41			p
ultrasonic	426	658				p
ultrasonic–closed room			7078			p
Paint						
during spray			161±30 mg m^{-3}			o
persistent			180±34 mg m^{-3}			o
			Radioactive aerosols			
None found						

* Except as noted

† Submicrometer particles only, background concentrations 5–24 μm g^{-1} m^{-3}

(a) Fradkin *et al* (1987), (b) Raes *et al* (1987) (c) Riley (1982) (d) Turiel (1985), (e) Langer and Fisher (1956), (f) Nelson *et al* (1988) (g) Dasch (1982), (h) Tu and Hinchliffe (1983), (i) Okada and Matsunuma (1974), (j) Burnet *et al* (1982), (k) Traynor *et al* (1982), (l) Girman *et al* (1982), (m) Mokler *et al* (1979a), (n) Hansen and Anderson (1986), (o) Mokler *et al* (1979b) (p) Highsmith *et al* (1988)

indoors by occupants Other types of these particles are produced indoors These particles do not present the infection potential of the animal aerosols but may be carcinogenic or mutagenic They also present problems by contaminating industrial environments and soiling furniture, etc These particles include asbestos, carbons, clays, elemental particles and artificial fibers

Asbestos, a major concern for building and health care professionals, is a carcinogenic fiber, formerly used in insulation Asbestos, as an indoor air contaminant, occurs when the coating over asbestos insulation degrades, releasing fibers into the air and during mitigation when asbestos is removed from buildings The first situation requires resealing or removal The removal procedure requires special containment apparatus Asbestos is an important source of indoor air pollution but is not an influential particle type in the choosing of ventilation and air-cleaning strategies since asbestos contamination must be addressed as a separate issue from routine indoor air quality

Talc is another source of mineral-based particles that is often indoor in origin These particles make up the majority of many types of body powders Thus, they are introduced into the air by the occupants at controllable intervals While many of these particles settle out of the air rapidly, others are inhaled especially since the powders are usually used close to or within the breathing zone

Man-made mineral fibers (MMMF) are used extensively in building materials, manufactured products and textiles They may be manufactured from ceramics, glass, rock, etc These fibers may enter with the outdoor air Indoors, MMMF can be generated in ventilation systems or when ceiling boards are damaged MMMF handlers have reported skin irritation, respiratory tract irritation and eye problems Other mineral particles vary greatly in size and shape, but need to be addressed in preventing their infiltration or extracting them from the air stream Again they may be inhaled if in the appropriate size range and can be a source of costly contamination to sensitive equipment

Combustion aerosols

Combustion aerosols are produced by burning Among the sources of this type of particle are cigarettes and other tobacco products, cooking sources, heating appliances and industrial plants These particles are produced in a gas stream and are lifted by the hot air into the surrounding environment Particles produced outdoors may enter through windows, doors, cracks or the HVAC system Most of these particles are in the respirable range and need to be taken into consideration when designing an air quality control system These sources are frequently considered for individual source venting, e g the chimney for the fireplace and the hood for the range These particles are a major source both of outdoor and indoor particles

Tobacco smoke, as the leading source of aerosol particles in smoking environments, is an extremely complex substance It contains particles and organic compounds The liquid particles and gases may condense on filters, then outgas later This substance is produced by smoking cigarettes, pipes, etc Tobacco smoke particles are almost all within the respirable range with the vast majority smaller than 1 μm Tobacco smoke creates allergy and odor problems The deposition of particles may require more frequent or extensive cleaning of drapes and furniture

Burning wood and other heating fuels, as well as cooking sources, also produce particles These particles are also mostly in the respirable range and should be considered in any IAQ control plan These sources are often difficult to eliminate as they are integral to the occupants' comfort and lifestyle

Industrial sources play a part even in indoor air These sources may pollute the ambient air in some regions to the extent that they contribute noticeably to the indoor particle counts In these cases tight controls on inlet air are important including filtration of the ambient air as it enters the HVAC system

Home/personal care aerosols

These products, including antiperspirants, dusting aids and hair sprays, are mostly sprays used in the home These products are designed to produce particles in order to deliver a product Note that in Tables 1 and 2 sizes are reported as a range of MMDs, therefore, the sizes in the distribution may vary considerably from these numbers These products produce a relatively small amount of mass but do so in a short period of time so that the initial concentration is quite high Since most of the use of these products is in the breathing zone, the dose delivered may be quite high One of the problems peculiar to this type of aerosol is that these products are often designed to stick to surfaces When this occurs to unintended surfaces, it often results in cleaning problems The smaller particles become part of the circulating air stream

Humidifiers are a relatively recent source of concern Research has shown that, along with the water droplets that are the intended output of humidifiers, these devices produce mineral and living aerosol particles Most of the minerals dissolved in tap water can become aerosol particles, possibly aggravating the health conditions that the units were intended to help Fungus and bacteria may grow in the units and become aerosols The vaporizer type of humidifier produces fewer of these living particles In addition to the health risks involved with both living and mineral aerosols, these particles may cause soiling of walls and floors Deionized, distilled or at least filtered water is recommended for use in these units

The other products in this category are sources found indoors after certain activities which can to some extent be controlled or at least monitored Still, all are considered necessary at times and present

health hazards by their chemical nature in addition to the particle size IAQ control strategies need to be adjusted whenever these sources are to be introduced

Radioactive aerosols

Radioactive aerosols are introduced into the indoor environment when radon enters through cracks in the basement or floor, in the water, and from exposed rock or sumps Radon decays to form radon progeny through nuclear degradation These particles are ultra small and may attach to larger particles

SUMMARY

Understanding indoor aerosols is important so that control techniques may be implemented to reduce damaging health effects and soiling problems A brief look at the mechanics of deposition in the lungs and on surfaces shows that particle diameters must be known to predict dose or soiling and to determine efficient mitigation devices Particle sizes produced by the various indoor sources, as well as unusual aspects of each type of source must be known so that this process may begin

There are several types of indoor particles plant and animal bioaerosols and mineral, combustion and home/personal care aerosols These types may be produced indoors or outdoors, entering through building openings The sources may be short term, seasonal or continuous Particle sizes produced vary from submicrometer to larger than 10 μm The particles may be toxic, allergenic or neutral All of these particles contribute to the indoor aerosol problem

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